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FOODBORNE ILLNESSES AND DEATHS

HEARING

BEFORE THE

COMMITTEE ON AGRICULTURE, NUTRITION, AND FORESTRY UNITED STATES SENATE

ONE HUNDREDTH CONGRESS

FIRST SESSION

JUNE 4, 1987

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CONTENTS

Hearing held on: Page
June 4, 1987 1

THURSDAY, JUNE 4, 1987

Leahy, Hon. Patrick J., a U.S. Senator from Vermont, opening statement 1
Wilson, Hon. Pete, a U.S. Senator from California 3
Pryor, Hon. David H., a U.S. Senator from Arkansas 4
Lugar, Hon. Richard G., a U.S. Senator from Indiana 5
Melcher, Hon. John, a U.S. Senator from Montana 5
Karnes, Hon. David K., a U.S. Senator from Nebraska 6
Cochran, Hon. Thad, a U.S. Senator from Mississippi, prepared statement 7
Heflin, Hon. Howell, a U.S. Senator from Alabama, prepared statement 8

CHRONOLOGICAL LIST OF WITNESSES

Bumpers, Hon. Dale, a U.S. Senator from Arkansas 10
Cohen, Mitchell L., M.D., Assistant Director for Medical Science, Division of Bacterial Diseases, Center for Infectious Diseases, Public Health Service, Department of Health and Human Services, Atlanta, GA 12
Prepared statement 14
Leighty, Jack C., D.V.M., Director, Pathology and Epidemiology Division, Science, Food Safety and Inspection Service, U.S. Department of Agriculture 25
Archer, Douglas L., Director of the Division of Microbiology, Center for Food Safety and Applied Nutrition, Food and Drug Administration, U.S. Department of Health and Human Services 41
Articles by Dr. Archer:
Incidence and Cost of Foodborne Diarrheal Disease in the United States 42
Enteric Microorganisms in Rheumatoid Diseases: Causative Agents and Possible Mechanisms 50
Roberts, Tanya, Agricultural Economics, Economic Research Service, U.S. Department of Agriculture 58
Prepared statement 61
Article, "The Economic Losses Due to Selected Foodborne Diseases" 69
Menning, Edward L., D.V.M., M.P.H., executive vice president, National Association of Federal Veterinarians 87
Prepared statement 90

MATERIAL SUBMITTED FOR THE RECORD

Rother, John C., director, legislation, research and public policy, American Association of Retired Persons [AARP], letter dated June 16, 1987, to Senator Patrick Leahy 108

FOODBORNE ILLNESSES AND DEATHS

THURSDAY, JUNE 4, 1987

U.S. SENATE,
COMMITTEE ON AGRICULTURE, NUTRITION, AND FORESTRY,
Washington, DC.

The committee met, pursuant to notice, at 9:45 a.m., in room 628, Dirksen Senate Office Building, Hon. Patrick J. Leahy (chairman of the committee) presiding.

Present or submitting a statement: Senators Leahy, Melcher, Pryor, Heflin, Lugar, Cochran, Wilson, and Karnes.

Also present: Senator Bumpers.

OPENING STATEMENT OF HON. PATRICK J. LEAHY, A U.S. SENATOR FROM VERMONT

The CHAIRMAN. The Committee on Agriculture, Nutrition, and Forestry will come to order.

We will be having a hearing today on foodborne illnesses and deaths. This is basically the hearing that we talked about some time ago. Let me just state briefly for the witnesses and for the committee our reason for this hearing.

The safety of our meat, poultry, and fish has received much public attention in the last few months. On March 27, I spoke on the Senate floor to express my serious concerns about the incidence of foodborne disease and illness. Since April, congressional committees have held three hearings on meat and poultry inspection. The National Research Council has issued a major report on inspection procedures. Secretary Lyng recently wrote to me that 3 to 5 percent of beef, 12 percent of raw pork, and 37 percent of broilers contain salmonella bacteria even after USDA inspects and approves the meat. The Centers for Disease Control, in a September 1985 study, stated that 24 percent of the food poisoning from meat, poultry, eggs, and seafood is caused by seafood alone. This figure is even more striking because seafood made up only 6 percent of meat consumption in 1985.

The Department of Agriculture has, in part, responded to the criticism. I noted that in their testimony before the House on Tuesday, they accepted the random sample concept proposed by the National Research Council. I am pleased to hear that, and I think that is a step forward. But they also indicated that they cannot afford to do the visual and random sample inspection. However, I feel strongly that they must do both. That is why I intend to develop new legislation addressing the problem, to begin to move beyond the cycle of hearing after hearing, of report after report, and move on to trying to correct the problem.

The current inspection system is "penny-wise, but pound-foolish." When you add up both the lost wages and medical expenses caused by foodborne illnesses, the total annual cost is in the billions. Thousands eventually die from complications from these diseases. We have to improve this situation quickly.

This committee is vitally interested in the wholesomeness of all foods. We held a full committee hearing last month on the problem of crop pesticide residues. It is a key role of Government to assure the safety of all our food supplies.

American farmers and ranchers produce the best meats, vegetables, dairy products, and poultry in the world. We need to ensure the high quality and nutritious content of these products when they get to the consumer. It is in the producers' and the processors' interest to have satisfied and healthy consumers.

However, many of the inspection procedures upon which we rely today have not been improved for decades. Visual inspections of the carcasses were described in Upton Sinclair's book, "The Jungle." That book was published in 1906. The current inspection system is like having surgery today with pre-World War I medical techniques.

It is clear that current inspection procedures fail to detect bacterial and chemical contamination of meat and poultry. With line speeds of 70 to 90 carcasses a minute, inspectors cannot catch anything more than gross contamination. But you cannot see the bacteria and chemicals that can make you sick. The potential problems associated with fish consumption are of equal concern. Because there is no mandatory inspection of fish, less than 20 percent of domestically produced fish consumed in the United States undergoes any type of inspection.

I plan to introduce comprehensive legislation which will address these problems at all levels of the food chain—from the feed which goes into the animals to the product opened in the consumer's kitchen. This bill will update our inspection procedures and help to restore public confidence in the safety of our red meats, fish, and poultry. I am hopeful that this legislation can set the stage for moving our inspection system into the 21st century and away from the turn of the century procedures we now have. The bill is still being drafted. I look for and will seek input from members of the committee and other interested parties. Many responsible industry associations already have study groups at work to come up with improvements. We will ask for their input.

Let me lay out the key points that I intend to include in this bill.

First, the bill will establish standards for what we feed our animals in order to reduce the chances of feeding them deadly bacteria or chemicals.

Second, at the slaughtering stage, we need to supplement the current inspection process with a random sample testing system to detect chemical residues and bacteria. And we need strict tolerance levels to ensure public safety and restore public confidence.

Third, the bill will create procedures to trace meat and poultry from the processor through to the retail meat counter. When we find a problem, we ought to know where it came from, and where it is going—a capability that does not exist now.

Fourth, at the final level of the food chain, the kitchen, the bill will establish a program to educate consumers on how to safely handle and prepare food products.

Fifth, the bill will establish mandatory Federal testing of fish using statistical sampling methods. Federal tolerance levels for dangerous chemicals, pesticides, and bacteria are will be established.

Finally, my bill will contain an employee protection system—a whistle-blower protection section—so that line inspectors can report improper practices without fear of reprisals by USDA or the plant where they are working. The inspectors are our first line defense against diseased meats.

I will actively seek comments as the bill is being drafted, and will hold hearings on the proposed legislation as it is completed. I look forward to working with other members of the committee on its development.

In that regard, I want to yield to the distinguished Senator from Arkansas who has done a great deal in this area and is one of the most knowledgeable members of this committee on this subject. Senator Pryor.

Senator PRYOR. Mr. Chairman, if I could yield at this time to Senator Wilson who has another committee he has to attend.

The CHAIRMAN. I yield to Senator Wilson from California.

STATEMENT OF HON. PETE WILSON, A U.S. SENATOR FROM CALIFORNIA

Senator WILSON. Thank you very much, Mr. Chairman, and I thank my friend from Arkansas for his great courtesy. I am required to be at the Commerce Committee markup momentarily.

Let me say that for years, I thought God had blessed me with a great tolerance for blue suits, and chicken, which made this calling natural, and beyond that, I even found that I have a particular favorite form of recreation. I like to barbecue chicken.

It is about the only culinary achievement I have, and it is a small one, but even that looks as though it may be imperiled.

In all seriousness, I commend the chairman for holding the hearing. It is particularly timely. I read a story in the June 3 issue of the Washington Post by Mr. Sinclair in which he reported a hearing on the House side, and without more than excerpting a small portion of the story, it refers to the testimony of J. Glenn Morris of the University of Maryland School of Medicine, who was quoted at the hearing as saying: "The reality is that 60 to 70 percent of the chickens that are being sent to market, may carry one or both of the common salmonella and campylobacter disease microorganisms."

The story goes on to paint a very grim picture, much on the order of what you referred to in your recollection of Upton Sinclair's book, "The Jungle," written in 1906, which portrayed the incredible conditions of the meatpacking industry at that time.

Mr. Chairman, I find the conditions that are portrayed in this report to be inexcusable.

Outrageous may not quite describe it, because what it indicates is that in the interest of stepping up production, which is by itself

commendable, that basic sanitation may be ignored at the great peril of the consumer. I will look forward to debating your legislation, and to other efforts, because it really seems to me that there was no excuse, in 1906, for the kind of situation that "The Jungle" portrayed.

But this is 1987, and there is absolutely no excuse, it seems to me, for subjecting American consumers to the peril that is portrayed in this report.

As I say, unless it is greatly exaggerated, it really demands attention and that it be cleared up.

The CHAIRMAN. Thank you, Senator.

Senator WILSON. Thank you, and I thank Senator Pryor.

The CHAIRMAN. Again I want to emphasize what I said in my initial statement, that I think America produces the best meats, fish, and poultry in the world. We just want to make sure that when the USDA stamp goes on, it says everything that consumers expect it too.

Senator Pryor.

STATEMENT OF DAVID H. PRYOR, A U.S SENATOR FROM ARKANSAS

Senator PRYOR. Well, Mr. Chairman, I did not have an opening statement. I do not have one prepared, and in fact I was not prepared to make an opening statement. I yielded my position to Senator Wilson and I am a little bit sorry I did, because I think Senator Wilson stated exactly what this hearing is not about.

This hearing is not about poultry. It is about all of the foods that we eat. It is about beef, it is about pork, it is about vegetables, it is about chocolates, it is about dairy, it is about cheese.

It is not about poultry alone. I think I can state, without any reservation, Mr. Chairman, that the poultry industry is the largest single employer today in the State of Arkansas. So you might say that Senator Bumpers and I have a conflict of interest.

So, when we try to single out one particular food, or to single out one particular process, I think that we have got to, in all fairness, put things in perspective, lay it all out on the table, and talk about the inspection system for all foods, all across the board.

For example, I am very proud, Mr. Chairman, that in your legislation that you have just announced a few moments ago, that you are going to propose that we have inspections for the fish industry.

Today, there is no inspection for the fish that we consume in our country. The Federal Food and Drug Adminstration is charged with that inspection process.

There is no process today. So, when we talk about poultry, let's talk about everything that we consume. Let's talk about everything that we buy at the grocery store, or eat in our restaurants, or cafes, or wherever. And all I am saying is, let us, certainly, Mr. Chairman, try to keep this hearing in perspective and not single out one particular problem without talking about the overall challenge that we, as a committee, and we, as a Congress, face. Thank you.

The CHAIRMAN. To quote a distinguished former Secretary of State, let me add the caveat that you are absolutely right. It is not just poultry. We are looking at all foodborne illnesses, and we are

looking at all areas where salmonella or other diseases—and we will discuss a number of them—can get into the food chain.

But more importantly, we are looking at those steps necessary to be able to guarantee to the American consumer that we will keep disease out of the food chain. And I think that is the most important thing.

I think it would be absolutely wrong to single out any one part of the food industry. Most people involved in the food industry in this country want to find ways to get all kinds of illnesses out, any kind of dangerous bacteria out, and that is what we will be looking at—how best to do it.

Senator Lugar.

STATEMENT OF HON. RICHARD G. LUGAR, A U.S. SENATOR FROM INDIANA

Senator LUGAR. Well, Mr. Chairman, I deeply appreciate your calling this hearing. I think the level of confidence of Americans in our food system will be enhanced by a historical review of those things which have been done by many gifted public servants over the years.

But likewise, this is a good time, as a benchmark oversight hearing, to see where we are now. The American people expect much more of us, and I am confident this hearing will lead to a great deal more understanding, and, hopefully, if we need legislation, enlightened legislation—if we do not—much better administrative authority. Thank you.

The Chairman. Thank you. I would also note for the committee that Senator Bumpers of Arkansas is here, too, and has asked if he might sit in on these hearings, part or all of them, and naturally we are delighted to have you here, Senator. He is a Senator who is extraordinarily knowledgeable in this area, and if you have anything you would care to say at this point, it is up to you.

Senator BUMPERS. Mr. Chairman, let me defer to the member of the committee, and then if you will, come back.

The CHAIRMAN. Thank you. And the member of the committee deferred to is Senator John Melcher, although I think in this case, we should refer to Senator Melcher as Dr. Melcher. He is, after all, a doctor of veterinary medicine, and the only doctor of veterinary medicine in the Senate, and I think perhaps in the Congress.

Senator MELCHER. That is true. Some people think it is one too many. [Laughter.]

The CHAIRMAN. Every time I hear about too many lawyers in Congress, John, I mention that we have a veterinarian in Congress, and there are those who suggest we would be better off with more veterinarians and less lawyers.

STATEMENT OF HON. JOHN MELCHER, A U.S. SENATOR FROM MONTANA

Senator MELCHER. That defense by yourself, counselor, is well taken.

The hearing you have called, Mr. Chairman, is a timely hearing. This is another one of those times when it has been proposed that meat and poultry inspection be paid by the plant, or the processor.

That is brought up from time to time, and is always defeated. The proposal never gets anywhere. We generally just look at it and say, well, whose idea is this, again?

Within the Department of Agriculture they are recommending something that we are going to have to take very seriously, and that is random inspection with more attention paid to possible bacterial infection in poultry.

I suspect that the public has become somewhat alarmed by recent media reporting of salmonellosis from poultry, or from other products, and to the extent that the inspection can be better, safer, and more reassuring of a wholesome product, I think we have the obligation here, in this committee, to consider legislation to implement it.

I am very pleased at the selection of witnesses. I see the leadoff group involves the Centers for Disease Control in Atlanta, and also, the Director of Pathology and Epidemiology for the Food Safety and Inspection Service.

I do not want to indicate that the second panel is any less auspicious, but I do believe, in this case, that the proper place to start is with the Centers for Disease Control, and the people responsible for the inspection service as it is now.

I am not only pleased, but I am also willing to participate in this hearing and to follow through on it, Mr. Chairman. I want to congratulate you for your timeliness in scheduling it, and your dedication to a clean, wholesome product, which has always been the trade of Patrick Leahy, counselor and Senator from Vermont.

The CHAIRMAN. I have to eat the food, too. I thank you very much, Senator Melcher, for those kind words, and at some point I will have to turn the gavel over to you anyway.

I yield now to Senator Karnes of Nebraska.

STATEMENT OF HON. DAVID K. KARNES, A U.S. SENATOR FROM NEBRASKA

Senator KARNES. Thank you, Mr. Chairman.

I want to commend your initiative on calling these hearings. We need to continually scrutinize our system of laws and regulations to assure that we are getting the safest possible food supply.

There are very few functions in Government which I can think of, that are of such a direct concern to each and every one of our citizens than the issue we address today.

But before I begin, I would like to make an observation. The meat industry has functioned very responsibly in the area of food safety. It is the most carefully regulated segment of the world's most carefully regulated food supply, and this is particularly applicable to the red meat industry, of which, being from Nebraska, I am most familiar.

Since the turn of the century, the Federal Government has maintained a constant presence in the industry reviewing the health of animals before and after slaughter, maintaining high standards of plant sanitation, and overseeing the full range of meat processing activities.

Due in fair measure to our success in eliminating some of the older problems such as many animal diseases, industry and Gov-

ernment are now turning their attention to some of the newer and more scientifically complex issues.

This is certainly appropriate, and as we learn more about topics such as the microbial safety of certain foods, it is important that we generate some innovative solutions to these problems.

I am sure that today's distinguished panel of witnesses will shed considerable light on these issues. I view such an effort as necessary and indicative of the sense of responsibility felt both by industry and by Government, to assure the safety and health of our citizenry.

We should also be wary of those who oppose such a change, be they in industry, Government, or labor, in order to simply protect the status quo for its own sake.

Clearly, we must demonstrate a continued willingness to change the system, to use new science and technology whenever it is appropriate, and to continue to do whatever we can to make the world's safest meat supply even safer.

Mr. Chairman, I have looked at the panelists today and I am very impressed with their credentials, and I look forward to participating in the hearing and hearing what they have to say. Thank you for the opportunity.

The CHAIRMAN. Thank you very much. Before we go to the panelists, Senator Cochran, did you have a statement?

Senator COCHRAN. Mr. Chairman, thank you.

I have a prepared statement for the record which I ask be inserted, and I look forward to hearing the testimony from the witnesses. Thank you.

The CHAIRMAN. It will be made a part of the record and also the prepared statement of Senator Heflin.

[The prepared statement of Senator Cochran follows:]

PREPARED STATEMENT OF SENATOR THAD COCHRAN, A U.S. SENATOR FROM MISSISSIPPI

HEARING ON FOODBORNE ILLNESSES

Mr. Chairman, I would like to take this opportunity to welcome the participants to this morning's hearing. I appreciate the fact that this hearing addresses the entire spectrum of foodborne illnesses from a scientific perspective.

I believe that it was Senator Pryor who suggested at the most recent FIFRA hearing that perhaps too many lawyers and not enough scientists have been addressing some of these issues. Today we will hear from several scientists, recognized in their respective fields. I hope that as you lend this committee the benefit of your collective expertise that we will be clearing up misconceptions and creating a record that will lead to useful decisionmaking.

It is generally recognized that here in the United States we have the safest food supply in the world. That did not happen by accident. That is not to say that there is no need for improvement. Today we will learn more about problem areas that merit our attention. As we seek a balanced, scientific perspective, we welcome your testimony.

[The prepared statement of Senator Heflin follows:]

OPENING STATEMENT
COMMITTEE ON AGRICULTURE, NUTRITION, AND FORESTRY
HEARING ON FOODBORNE ILLNESSES IN THE UNITED STATES

MR. CHAIRMAN, I WOULD LIKE TO COMMEND YOU FOR YOUR DILIGENT WORK IN HOLDING THIS HEARING ON THE ISSUE OF FOODBORNE ILLNESSES.

OVER THE PAST SEVERAL WEEKS, THIS ISSUE HAS RECEIVED A TREMENDOUS AMOUNT OF ATTENTION AND PUBLICITY. IT IS AN ISSUE THAT AT TIMES CAN BECOME VERY EMOTIONAL. THIS HEARING CAN BE VERY BENEFICIAL TO THIS COMMITTEE, TO CONGRESS AND TO THE AMERICAN PUBLIC. I WOULD HOPE THAT THESE HEARINGS WILL SERVE AS A FORUM FOR OBTAINING FACTS ON THE GENERAL ISSUE AND NOT A PLACE FOR SINGLING OUT ONE INDUSTRY AND FURTHERING CONSUMER FEAR.

MR. CHAIRMAN, OUR ROLE IS TO TRY TO RESTORE ANY LOSS OF PUBLIC CONFIDENCE IN THE AMERICAN FOOD PROCESSING INDUSTRY DUE TO RECENT NEWS REPORTS AND OTHER ATTENTION. THE AMERICAN CONSUMER DESERVES TO KNOW IF THERE IS A REASON TO BE CONCERNED WITH THE SAFETY OF THIS COUNTRY'S FOOD SOURCE.

THERE ARE CERTAIN QUESTIONS THAT I FEEL WE HAVE TO ANSWER. IS THERE A PROBLEM IN THE INSPECTION FOR SUCH DISEASES AS SALMONELLOSIS? OR IS THIS AS MUCH A PROBLEM WITH

CONSUMER MISHANDLING OF POULTRY, MEAT AND OTHER FOOD PRODUCTS? IT IS MY UNDERSTANDING THAT IF THESE PRODUCTS ARE COOKED PROPERLY THIS BACTERIA OR DISEASE ORGANISM IS DESTROYED. SHOULD THERE BE MORE CONSUMER EDUCATION IN THIS AREA? ARE THESE SORTS OF DISEASE BACTERIA ANY MORE PREVALENT TODAY THAN THEY WERE TEN YEARS AGO? IS THERE A COUNTRY IN THE WORLD THAT HAS A FINER SYSTEM OF FOOD PROCESSING AND INSPECTION?

OUR FOOD PROCESSORS AND SUPPLIERS HAVE MADE GREAT ADVANCES IN PROVIDING AND PROMOTING SAFE CLEAN FOOD PROCESSING OVER THE PAST SEVERAL YEARS. AND THEY HAVE DEVELOPED A MODEL FOR THE REST OF THE WORLD IN THEIR COMMITMENT TO QUALITY PRODUCTS, IN A SAFE MARKET PLACE.

MR. CHAIRMAN, I HOPE THE COMMITTEE WILL ENDEAVOR TO OBTAIN ANSWERS TO SUCH QUESTIONS. I, JUST LIKE MY COLLEAGUES, WANT THE SAFEST POSSIBLE FOOD SUPPLY FOR THE AMERICAN PUBLIC, BUT I WANT TO BE SURE WE HAVE THE NECESSARY INFORMATION BEFORE WE PROCEED WITH LEGISLATIVE MANDATES.

THANK YOU MR. CHAIRMAN.

The CHAIRMAN. Senator Bumpers, as we said earlier, if you want to say something, please feel free to do so.

STATEMENT OF HON. DALE BUMPERS, A U.S. SENATOR FROM ARKANSAS

Senator BUMPERS. Mr. Chairman, first, let me thank you for allowing me to sit with the committee this morning. As Senator Pryor has already pointed out, this hearing—and thanks to the chairman—is not just about poultry.

It is about the food safety inspection system, and whether or not the Department of Agriculture is doing all it can to protect the health of the American people.

But the genesis of this hearing is the show that "Sixty Minutes" did a couple of months ago, which I watched with a great deal of interest, knowing that it was going to be coming on. And as usual, of course, as they say, they "did a number" on the poultry industry, and that is a good way to get Senator Pryor's and my attention, not because we have a conflict of interest but because we have an interest.

I remember one Senator who used to say he did not support the B–1 bomber because the engines were made in Ohio, even though he did happen to be a Senator from Ohio.

And so, when you consider the fact that 1 out of every 12 people in my State is employed in the poultry industry, of course we have an interest. We do not have an interest, however, in protecting an industry that is putting out an unwholesome product. On the contrary, our interest is to make sure that the industry puts out a wholesome product. Who do you think has the greatest interest in making sure that poultry is not only consumed in this country but consumed as a healthy, wholesome product? It is the industry.

What profiteth it for an industry to put out an unwholesome product, to generate a story like the one on "60 Minutes"? The answer is nobody would have a greater interest than the industry, to make certain that stories like that do not come out.

To suggest that poultry is the only culprit is palpably unfair, and in addition to that, if you remember the "60 Minutes" story, not one single person who had been ill with salmonella from eating poultry was ever shown on that program. Not one.

To say that the incidence of salmonella from poultry consumption is up is certainly a credible statement. The first year I was Governor of Arkansas, in 1971, the people of this country were consuming in the neighborhood of 17 pounds of poultry per capita per year, and about 110 pounds of beef per year.

This year, 1987, consumers in this country will eat 74 pounds of poultry and 74 pounds of beef. It will be even. And we all know that the reason for the dramatically increased consumption of poultry and fish in this country is because of health reasons.

The point I want to make is, obviously, when you consider that the consumption of poultry is 500 times greater now than it was 15 years ago, it is quite obvious that there would be some increased incidence.

Dr. Houston would testify that 35 percent of all the poultry in this country contains salmonella when it comes off the assembly

line, and that is precisely what it was in the years 1965 through 1975.

When you go back and you look at CDC figures, which incidentally, CDC and HHS will both tell you that their figures are not really as conclusive and as definitive as they would like for them to be, through 1982, and from 1966 through 1975, there were 500 outbreaks in the country; 6 percent of which were due to poultry consumption, out of 500 outbreaks.

I am not talking about individual cases. There were obviously thousands of cases. I am talking about outbreaks. If you look closer at those statistics, you will find that almost invariably, the outbreak occurred because of consumption at an institution, a college dining room, a restaurant, a hospital, not home consumption.

In 1978, there were 51 outbreaks, 4 percent due to poultry. In 1979, 67 outbreaks, 4.5 percent due to poultry. In 1980, 39 outbreaks, none due to poultry. And the figures go on like that.

So I am not going to bore you with all the statistics that I have from both HHS, HEW—its predecessor—CDC, and so on, but those of us who have a deep and abiding interest in the viability of the poultry industry want to make certain that they are not unfairly accused, and if they are accused that it is fair, and that the remedies—if any—that are in order, that this committee might choose to recommend to the Congress, are remedies that are going to deal with the problem and not be discriminatory.

In conclusion I have been sick from food poisoning a few times in my life, but not once from eating poultry.

I can tell you I have been sick many times, and every time, except two that I can remember, it was from eating seafood, which is not federally inspected. I went to that seafood luncheon, and I guess this is really——

The CHAIRMAN. Now, now, now, we will not get invited next year.

Senator BUMPERS. I will not go into it. You got the message quickly, Mr. Chairman. But as you know, seafoods are not federally inspected in this country, and that is what has always caused my food poisoning, plus an occasional dairy product, and yet I am not——

[Laughter.]

Senator BUMPERS. I guess I had better quit, Mr. Chairman.

The CHAIRMAN. Boy, I tell you, we were just about to change the witness list in a heck of a hurry here, after that remark.

Senator PRYOR. I think the chairman really got the message on that, Senator Bumpers.

Senator BUMPERS. I do not want to seem like an ingrate, Mr. Chairman.

The CHAIRMAN. As I said in my opening statement we are looking at all foods—red meat, poultry, fish, and dairy products.

For those who, after hearing about these hearings, decide to become total vegetarians, I would ask them to go back and read the transcript of the FIFRA hearings that we held just a week or two ago.

Sometimes one of the problems with being on this committee is that it is like being a first-year medical or law student. If you are a first-year medical student, you are convinced you have every dis-

ease you are going to read about. If you are a first-year law student, you wonder how you ever stayed out of prison during the time you were doing your undergraduate work.

We are looking for ways not to condemn any industry, but to find out how to make the industry safer. This is the mandate I think most consumers give to the Department of Agriculture and it is our responsibility as the oversight committee to ensure that.

Incidentally, for the record, on fish, in 1985, 13 percent was inspected, and that I believe is a voluntary inspection procedure.

So, we have said enough. Let's hear from the experts. Dr. Cohen and Dr. Leighty, if you would come forward and join us at the table.

Now, gentlemen, who is starting first? Dr. Cohen.

STATEMENT OF MITCHELL L. COHEN, M.D., ASSISTANT DIRECTOR FOR MEDICAL SCIENCE, DIVISION OF BACTERIAL DISEASES, CENTER FOR INFECTIOUS DISEASES, PUBLIC HEALTH SERVICE, DEPARTMENT OF HEALTH AND HUMAN SERVICES, CENTERS FOR DISEASE CONTROL, ATLANTA, GA

Dr. COHEN. Thank you. I am Mitchell Cohen, Assistant Director for Medical Science, Division of Bacterial Diseases [DBD], Center for Infectious Diseases [CID], Centers for Disease Control [CDC]. I am pleased to respond to the committee's invitation to discuss foodborne diseases in the United States.

I have a prepared statement that I would like to request be inserted into the record, and I would like to make a brief statement to emphasize a few of the important points which are in the prepared statement.

The CHAIRMAN. Certainly.

Dr. COHEN. Foodborne disease surveillance in the United States is the collection, analysis, and dissemination of data on foodborne illness. Surveillance has been going on for over 50 years. CDC assumed the responsibility for national foodborne disease surveillance in the 1960's.

CDC has several sources of foodborne disease data. The primary sources of data are the reports of outbreaks of foodborne disease that are submitted by State and local health departments. CDC also conducts surveillance of laboratory confirmed cases of specific diseases, such as salmonellosis, which may be transmitted by foods.

CDC also directly participates in investigation of certain disease outbreaks, and conducts studies of specific public health problems.

The information developed through foodborne disease surveillance has traditionally had three objectives.

The first is to assist disease prevention and control.

The second is to broaden knowledge of disease causation.

The third is to provide administrative guidance.

To appropriately use foodborne disease data to accomplish these objectives, it is important to appreciate the limitations that are inherent in outbreak data. The first limitation is that the number of outbreaks of foodborne disease that are reported by the surveillance system represents only a fraction of those cases that occur in the country each year. In a sense, the reported cases are really only the "tip of the iceberg." For an outbreak to be counted, it

must be investigated by a health authority, a questionnaire completed and submitted to CDC.

The second limitation of using data from reported outbreaks is that these outbreaks are not a random sample of all cases of foodborne disease. There are reasons why some outbreaks are more likely to be detected and reported than other outbreaks.

Similarly, a food's importance as a cause of disease outbreaks may not be proportional to its true incidence as a cause of disease. The data in this statement are primarily obtained from the analysis of outbreak data.

Outbreaks are defined as episodes in which illness involves two or more persons. Some foodborne disease primarily affect individuals which are not part of any recognized cluster or outbreak. Such ill persons are frequently referred to as "sporadic cases" of disease. The foods which are important causes of outbreaks may or may not be important causes of disease in these sporadic cases.

Thus, these data should not lead to firm conclusions about the absolute incidence of foodborne disease. They should not be used to draw conclusions about the relative incidence of specific causes or specific foods. These data are perhaps most useful in examining trends, or changes over time.

When we review the data on outbreaks of foodborne diseases from 1973 to 1984, there are three major conclusions. The first is that foodborne disease is an important public health problem, which has been estimated to affect over 6 million Americans each year. Second, the number of reported outbreaks is increasing, and this increase is primarily attributable to an increase in the number of salmonellosis outbreaks. And finally, most vehicles of foodborne disease, regardless of the pathogen that they transmit, are of, or contain, foods of animal origin.

I would be pleased to discuss any of the information which is presented in the prepared statement or answer any questions. Thank you.

[The prepared statement of Dr. Cohen follows:]

DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Centers for Disease Control
Atlanta GA 30333

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I am Mitchell Cohen, Assistant Director for Medical Science, Division of Bacterial Diseases (DBD), Center for Infectious Diseases (CID), Centers for Disease Control (CDC). I am pleased to respond to the Committee's invitation to discuss foodborne diseases in the United States. I am accompanied by Dr. Douglas L. Archer, Director of the Division of Microbiology, Center for Food Safety and Applied Nutrition, Food and Drug Administration (FDA). Dr. Archer will be available to answer questions on the etiology of foodborne illnesses.

Reporting of foodborne diseases in the United States began over half a century ago when State and territorial health officers, concerned about the high morbidity and mortality caused by typhoid fever and infantile diarrhea, recommended that cases of enteric fever be investigated and reported. The purpose was to obtain information about the role of food, milk, and water in outbreaks of intestinal illness as the basis for sound public health action. Beginning in 1923, the U.S. Public Health Service published summaries of outbreaks of gastrointestinal illness attributed to milk. In 1938, it added summaries of outbreaks caused by all foods. These early surveillance efforts led to enactment by regulatory agencies of important public health measures which had a profound influence in decreasing the incidence of enteric diseases, particularly those transmitted by milk and water.

From 1951 through 1960, the National Office of Vital Statistics reviewed reports of outbreaks of foodborne illness and published summaries of them annually in Public Health Reports. In 1961 the Centers for Disease Control, then the Communicable Disease Center, assumed responsibility for publishing reports on foodborne illness.

In 1966 the present system of surveillance of foodborne diseases began with incorporation of all reports of enteric disease outbreaks attributed to microbial or chemical contamination of food or water into an annual summary. Since 1966 the quality of investigative reports has improved as State and federal agencies have participated more actively in the investigation of foodborne disease outbreaks.

Foodborne disease surveillance is the collection, analysis, and dissemination of data on foodborne illness. CDC has several sources of these data. In addition to receiving reports of outbreaks of foodborne diseases from State and local health departments, CDC conducts surveillance of cases of specific diseases that may be transmitted by foods such as salmonellosis. CDC also investigates certain disease outbreaks and studies specific public health problems.

Foodborne disease surveillance has traditionally had 3 objectives:

1. Disease Prevention and Control: Surveillance data can lead to early identification and removal of contaminated products from the commercial market, correction of faulty food preparation practices in food service establishments and in the home, and identification and appropriate treatment of human carriers of foodborne pathogens. These are the fundamental prevention and control measures resulting from surveillance of foodborne diseases.

2. Knowledge of Disease Causation: More thorough clinical, epidemiologic, and laboratory investigations can identify many of the pathogens responsible for outbreaks and can lead to suitable measures for prevention and control. This is important because the responsible pathogen was not identified in over half of the foodborne disease outbreaks reported to CDC. In many of these outbreaks pathogens known to cause foodborne illness may not have been identified because of late or incomplete laboratory investigation. In others, the responsible pathogen may have escaped detection even when a thorough laboratory investigation was carried out because the pathogen could not be identified by available laboratory techniques or because the pathogen was not a known cause of foodborne disease. Such is the case for Campylobacter jejuni, Escherichia coli 0157:H7, and Listeria monocytogenes which have only been recognized as foodborne pathogens in the last several years.
3. Administrative Guidance: The data collected by surveillance can guide health departments, regulatory agencies, and industry in planning long-term efforts to reduce foodborne illness.

The limitations on the quantity and quality of foodborne data must be appreciated to avoid misinterpretation. The number of outbreaks of foodborne diseases reported by the surveillance system clearly represents only a small fraction of the total number that occur. The likelihood of an outbreak coming

to the attention of health authorities varies considerably depending on consumer and physician awareness, interest, and motivation to report the incident; for example, large outbreaks, interstate outbreaks, restaurant-associated outbreaks, and outbreaks involving serious illness, hospitalizations, or deaths are more likely to come to the attention of health authorities than are cases of mild illness following a family cookout.

The quality of the data depends upon the State or local health departments' interest in foodborne diseases and its investigative and laboratory capabilities. The likelihood that the findings will be reported varies from one locality to another. Thus, these data should not lead to firm conclusions about the absolute incidence of foodborne diseases, and they should not be used to draw conclusions about the relative incidence of foodborne diseases of various etiologies. For example, foodborne diseases characterized by short incubation periods, such as those of chemical etiology or outbreaks caused by staphylococcal enterotoxin, are more likely to be recognized as common-source foodborne disease outbreaks than are those diseases with longer incubation periods, such as hepatitis A. Outbreaks involving less common or more difficult to culture pathogens, such as Bacillus cereus, Escherichia coli, Vibrio parahaemolyticus, Yersinia enterocolitica, or Campylobacter jejuni are less likely to be confirmed because these organisms are often not considered in clinical, epidemiologic, and laboratory investigations. Pathogens which generally cause mild illness will be

under-represented, while those causing serious illness, such as Clostridium botulinum, are more likely to be identified. Similarly, foods which are served to greater numbers of persons or restaurant- or commercial product-associated outbreaks have a higher likelihood of being detected and reported.

Outbreak data may not be representative of all reported cases. In 1983, 38,881 Salmonella infections were reported to CDC by the Salmonella surveillance system. In contrast, the foodborne surveillance system only reported a total of 2,047 cases as outbreak-associated. There may be important differences between the epidemiology of outbreaks of foodborne diseases and that of sporadic cases of disease that are not recognized as being part of any outbreak. For example, several studies indicate that Campylobacter may be the most common bacterial cause of diarrheal disease in the United States, but it is an uncommon cause of foodborne disease outbreaks. Of the small number of reported Campylobacter outbreaks, almost two-thirds have been associated with dairy products, whereas over one-half of sporadic cases have been associated with poultry consumption.

Keeping in mind the limitations of these data, we can examine the trends in foodborne diseases for the period 1973 through 1984.

During the study period, there were 5,924 reports of outbreaks affecting 171,072 persons. The total number of outbreaks, cases, and deaths over the 12 years is shown in Table 1. In 2,320, or 39%, outbreaks involving 86,121 cases, an etiology was confirmed. These etiologies can be grouped into bacterial, chemical, parasitic, and viral. Bacterial pathogens accounted for

69% of confirmed outbreaks and 85% of cases. Salmonella was the pathogen most frequently responsible for outbreaks, followed by S. aureus and C. perfringens (Table 2).

Although the total number of reported outbreaks has gradually increased from 1973 to 1984, changes in the numbers of bacterial and parasitic outbreaks are particularly striking (Figure 1). The increase in bacterial outbreaks is primarily related to an increase in Salmonella outbreaks (Figure 2). The decrease in parasitic outbreaks is attributable to a decrease in trichnosis.

There were 202 deaths associated with foodborne diseases. Outbreaks of C. botulinum intoxication and Salmonella infections accounted for 48% of deaths.

It is important to note that for certain diseases, both the reported cases and deaths are only a fraction of the numbers of cases estimated to actually occur. For example, although 40,000 cases of Salmonella infection are reported annually, several studies indicate that between 400,000 and 4,000,000 cases and over 500 deaths occur each year in the United States. Additional studies suggest that over 90% of salmonellosis cases are foodborne.

A number of different vehicles were implicated in the outbreaks reported between 1973 and 1984 (Table 3). For most foods, the number of outbreaks per year show no consistent trend (Table 4). For specific pathogens, the most common vehicles are shown in Table 5. Most of the increase in outbreaks of salmonellosis was from undetermined vehicles (Table 6). For beef, changes in reported numbers of outbreaks correspond to recognition and resolution of problems encountered in the mid- and late 70's with precooked roast beef.

Most vehicles of foodborne diseases are or contain foods of animal origin. These foods are typically high protein, support bacterial growth, and may be intrinsically contaminated. As has been recognized for many years, the critical elements in foodborne diseases are the presence of pathogens in the food and the food handling conditions that allow the pathogen to survive or replicate.

Thank you for the opportunity to discuss foodborne disease. I will be happy to answer questions you or members of the Committee may have.

Table 1 Epidemiologic Characteristics of Reported Outbreaks of Foodborne Disease by Etiologic Group: United States, 1973-84.

Etiologic Group	Number of Outbreaks	Cases	Deaths	Mean Outbreak Size	Death-to-Case Ratio*
Bacterial	1591	73359	152	46.1	2.1
Parasitic	106	795	4	7.5	5.0
Viral	105	8425	2	80.2	0.2
Chemical	518	3542	19	6.8	5.4
Unknown	3604	84946	25	23.6	0.3
Total	5924	171067	202	28.9	1.2

*Deaths per 1000 cases

Table 2 Epidemiologic Characteristics of Reported Outbreaks of Foodborne Disease Caused by Specific Bacterial Pathogens: United States, 1973-84.

Pathogen	Number of Outbreaks	Cases	Deaths	Mean Outbreak Size	Death-to-Case Ratio*
Salmonella	597	30296	60	50.7	2.0
Staphylococcus aureus	358	17332	4	48.4	0.2
Clostridium perfringens	214	12432	12	58.1	1.0
Clostridium botulinum	183	428	37	2.3	86.5
Shigella	78	5647	2	72.4	0.4
Bacillus cereus	53	777	0	14.7	0
Campylobacter**	37	1289	2	34.9	1.6

*Deaths per 1000 cases
**First reported in 1978

Table 3 Number of Reported Outbreaks of Foodborne Disease by Etiologic Group and Vehicle, United States, 1973-84.

Vehicle	Etiologic Group					
	Bacterial	Parasitic	Viral	Chemical	Unknown	Total
Beef/Veal	159	8	0	2	139	308
Pork/Ham/Sausage	143	71	3	2	68	287
Lamb	3	0	0	0	3	6
Chicken	47	0	2	0	49	98
Turkey	79	0	1	0	49	129
Fish	44	7	2	280	41	374
Shellfish	33	0	12	21	114	180
Dairy Products	70	0	2	2	33	107
Mixed Vehicles and Other (incl. eggs)	634	15	38	205	617	1509
Unknown	379	5	45	6	2491	2926
Total	1591	106	105	518	3604	5924

Table 4 Number of Reported Outbreaks of Bacterial Foodborne Disease by Vehicle, United States, 1973-1984.

Vehicle	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	Total
Beef/Veal	9	8	19	8	12	10	17	13	22	16	9	16	159
Pork/Ham/Sausage	7	16	21	4	12	18	12	9	16	9	9	10	143
Lamb	1	0	0	0	0	0	0	1	0	0	1	0	3
Chicken	3	1	3	4	3	1	5	3	13	6	4	1	47
Turkey	8	3	4	3	4	3	2	15	14	11	6	6	79
Fish	6	7	5	2	1	2	3	3	3	3	4	5	44
Shellfish	1	0	1	0	3	6	6	5	4	4	1	2	33
Dairy Products	2	4	1	2	2	3	1	4	11	9	17	4	70
Mixed Vehicles and Other (incl. eggs)	39	59	53	49	52	56	53	66	52	60	52	53	634
Unknown	8	24	18	20	14	31	35	18	53	44	36	78	379
Total	84	122	125	92	103	130	134	137	188	162	139	175	1591

Table 5 Number of Reported Outbreaks of Foodborne Disease Caused by Specific Bacterial Pathogens by Vehicle, United States, 1973-84.

	Bacterial Pathogen						
Vehicle	Salmonella	S. aureus	C. perfringens	C. botulinum	Shigella	B. cereus	Campylobacter*
Beef/Veal	70	21	60	1	0	3	0
Pork/Ham/Sausage	25	106	11	0	0	0	0
Lamb	1	2	0	0	0	0	0
Chicken	19	14	8	1	1	1	1
Turkey	33	19	22	1	1	2	1
Fish	5	4	4	20	5	1	0
Shellfish	2	2	2	0	2	1	0
Dairy Products	21	6	0	0	0	0	22
Mixed Vehicles and Other (incl. eggs)	190	160	68	131	34	40	3
Unknown	231	24	39	29	35	5	10
Total	597	358	214	183	78	53	37

*First Reported in 1978.

Table 6 Number of Reported Outbreaks of Foodborne Salmonellosis by Vehicle, by 3 Year Periods, United States, 1973-84.

Vehicle	'73-'75	'76-'78	'79-'81	'82-'84	Total
Beef/Veal	13	20	24	13	70
Pork/Ham/Sausage	4	8	6	7	25
Lamb	1	0	0	0	1
Chicken	2	5	6	6	19
Turkey	6	7	12	8	33
Fish	2	0	0	3	5
Shellfish	0	0	2	0	2
Dairy	3	5	8	5	21
Mixed Vehicles and Other (incl. eggs)	50	47	42	51	190
Unknown	29	36	55	111	231
Total	110	128	155	204	597

FIGURE 1: NUMBER OF REPORTED OUTBREAKS OF FOODBORNE DISEASE BY ETIOLOGICAL GROUP AND YEAR, U.S. 1973–1984

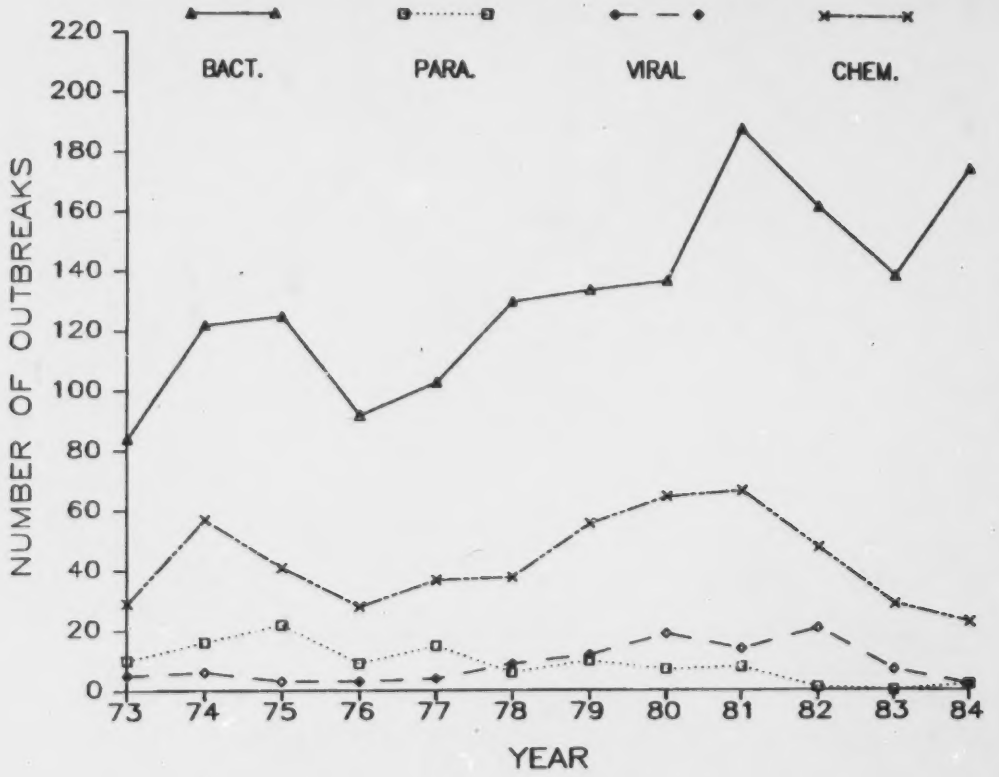
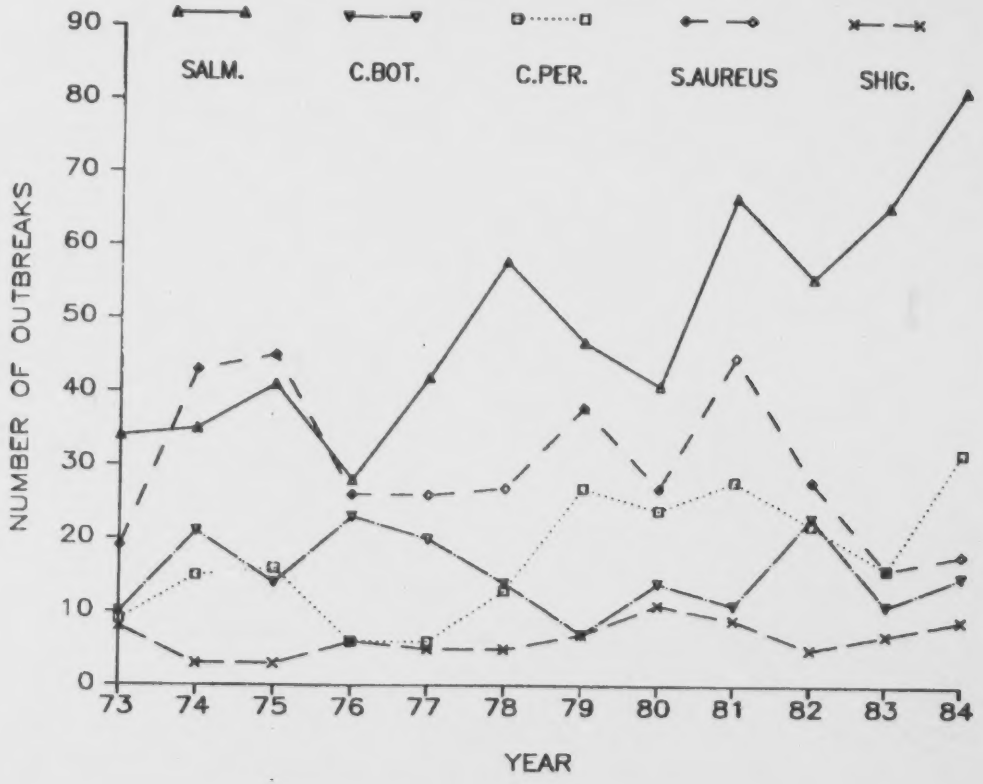


FIGURE 2: NUMBER OF OUTBREAKS OF BACTERIAL FOODBORNE DISEASE BY PATHOGEN AND YEAR, U.S. 1973–1984



The CHAIRMAN. Thank you very much, and before we begin the questions, Dr. Cohen, Dr. Leighty, your testimony, please.

STATEMENT OF JACK C. LEIGHTY, D.V.M., DIRECTOR, PATHOLOGY AND EPIDEMIOLOGY DIVISION, SCIENCE, FOOD SAFETY AND INSPECTION SERVICE, U.S. DEPARTMENT OF AGRICULTURE

Dr. LEIGHTY. I am Jack Leighty. I am Director of the Pathology and Epidemiology Division of the Food Safety and Inspection Service, USDA.

Domesticated food animals, the avian and mammalian species from which our meat supply is derived, are affected by an enormous variety of disease conditions, having an equally great and complex number of causes. Of greatest significance to human health are the zoonotic diseases. These are the infectious diseases that can infect humans as well as one or more species of food animal. However, even those disease conditions that affect only food animals often have human health implications. Sick animals are more susceptible to, and are likely to have their primary disease condition complicated by, infectious agents that do cause human illness.

Most food animals are found to be healthy at the time that they enter the meat supply. The year 1985 was typical with respect to the number of food animals that arrived at abattoirs. This included 4.8 billion chickens, turkeys and other domesticated fowl; 33.3 million cattle; 5.9 million sheep and goats; and 78 million swine. Each of these animals was individually inspected, and relatively few were found to be diseased or otherwise unfit for food. Nevertheless, even those low condemnation rates accounted for rather large numbers of condemned animals. For example, although only 0.98 percent of the poultry was condemned at post mortem inspection, 47.3 million unfit carcasses were discarded. The total weight of the poultry condemned, that year, at both ante mortem and post mortem inspection, was approximately 272 million pounds.

The data on other species are similar. Percent condemnation for cattle in 1985 was 0.37; for sheep and goats 0.52; and for swine 0.23 percent. Condemnation rates for these species have changed little over the past 20 years.

We have a great deal of knowledge regarding the zoonotic diseases of food animals. However, there is still much, of great importance to both human and animal health, about which we have little knowledge, and the required knowledge is difficult to acquire. For example, while ante mortem and post mortem inspection is required for each animal that is to become meat, the inspection produces little data of value to animal disease control. The currently utilized inspection procedures are limited largely to observation. The inspecting veterinarian and assistants see the signs, symptoms and lesions of many different disease conditions that are seldom pathognomonic, that is, characteristic of only one disease specific for that particular disease condition. The large numbers of animals involved, the usual absence of a credible health history, and the impracticality of routine laboratory assistance make a specific diagnosis of each observed disease condition impossible.

To further complicate this picture, apparently healthy animals are sometimes carriers of dangerous infectious or toxic agents that can be passed through their meat to consumers. These may be organisms, such as brucella, toxoplasma, or trichina, which persist in the animal's tissues after the animal recovers from the disease; or may be residues of toxic chemicals, such as industrial wastes, pesticides, or antibiotics. Finally, apparently clean meat may become contaminated during slaughtering and processing with hazardous microbes or chemicals.

Rapid testing systems are being developed which may, in the future, be of great value in obtaining a specific diagnosis of a limited number of diseases that are of particular significance to human and animal health. However, it would be naive to believe that such tests will enable either a specific diagnosis of each disease with which the inspector may be confronted, or a complete evaluation of the suitability of the animals for food.

The inability of the veterinary inspector to make a specific diagnosis of all diseases encountered is of little practical significance to the essential task of identifying diseased animals and removing them from the food supply at the abattoir. However, it does limit our epidemiological knowledge of factors, such as incidence and prevalence, that are of great importance to zoonotic disease control. Data collected by production oriented animal disease reporting systems are of great value, but are also deficient, in many respects, in their contribution to our knowledge of zoonotic disease.

A similar problem exists on the human side. The zoonotic diseases produce syndromes in humans that are also confusingly similar to those of other diseases. In many cases, a physician's assistance is not sought by the person affected, or, for other reasons a specific diagnosis is not made by the physician. And often, when a disease is specifically identified, that fact is not reported in a manner that makes it a useful part of our knowledge of the disease. The deficiencies in the production of important human and animal disease data increase the difficulty of developing effective strategies for controlling meatborne diseases, and of measuring the progress of control programs.

A brief description of some of the infectious agents capable of causing human meatborne illness may provide some appreciation of the complexity of the meat hygiene problem.

The most common bacterial organisms that may be transmitted to humans through the handling or consumption of meat are Bacillus anthracis, Brucella, Corynebacteria, Erysipelothrix, Pasteurella, Staphylococcus, Streptococcus, Listeria, and the enteric disease organisms, Salmonella, Yersinia, Campylobacter, enteropathogenic Escherichia coli, and Clostridia.

The diseases caused by the Actinomyces include tuberculosis, actinomycosis, and nocardiosis.

The Rickettsia cause chlamydiosis—which you may recognize as psittacosis, ornithosis—and Q fever. The chlamydial infections of humans often result in significant heart damage that persists after the acute phase of the disease.

The only known spirochaete disease, of importance, is leptospirosis. This organism occurs in all tissues during the acute phase of the disease, then often chronically infects the kidneys.

Trichinosis is the most common nematode caused disease. However, others occur rarely when inadequately cooked, contaminated meat is consumed. Highly effective logical tests, which have recently become available, have the potential of controlling this disease almost to the point of eradication, if properly utilized.

The cestodes of importance include the beef tapeworm, Taenia saginata, and the pork tapeworm, Taenia solium.

By far the most important protozoan infection, transmitted to humans by consumption of meat from infected animals, is toxoplasmosis. Where consumption of rare or uncooked meat is common, serological studies indicate a high prevalence of human infection. In Paris, for example, a prevalence of 90 percent has been reported in women. Toxoplasmosis is a significant cause of birth defects. Control of the disease in the definitive host, the cat, by vaccination would largely eliminate the infection hazard for both food animals and humans. A second protozoan agent, Crytosporidia, is emerging in importance as cause of human enteric disease, as our knowledge of the biology and epidemiology of the organism grows.

Rabies occurs in all food animals. It is an important consideration when inspecting animals for evidence of encephalitis at ante mortem. Newcastle disease of poultry, also caused by a virus, causes eye infections, and rarely, encephalitis in humans.

In summary, the many diseases that may be transmitted to humans through meat and poultry constitute a problem that requires the continuous efforts of medical professionals if it is to be effectively controlled.

The CHAIRMAN. Thank you very much.

Dr. Cohen, when I read your article in the "Science" magazine on drug resistant salmonella in the United States, you, as I understand it, explained that farmers feed their animals antibiotics as a growth enhancer, is that correct?

Dr. COHEN. Yes. That is correct.

The CHAIRMAN. And that antibiotics actually increase growth?

Dr. COHEN. The antibiotics are used for several purposes—including growth promotion, and disease prevention.

The studies, which examined the efficacy of growth promotion, date back to the 1950's, and may have a strong element of disease prevention.

I am not sure that the "growth promoting" effect is always predictable, but my area of expertise is the impact of antibiotic resistance on human health.

The CHAIRMAN. Have other countries banned the use of antibiotics as a growth enhancer in animals because they fear that human health may be adversely affected?

Dr. COHEN. Yes.

The CHAIRMAN. Would you recommend such a ban in the United States?

Dr. COHEN. I think we have to look at how we use antimicrobials in animals in general. Subtherapeutic use may contribute to disease, but documented public health problems have also arisen from the therapeutic use of antimicrobials in feeds, or through injectables. So that I think we have to address the entire spectrum of antimicrobial use in animals.

The CHAIRMAN. What percentage of foodborne illnesses and deaths are attributable to seafood?

Dr. COHEN. The only data are from outbreak investigations. It is very difficult to say whether those outbreak data are a true representation of the seafood related illness for several reasons.

One, seafood associated diseases may be more readily detected because of the nature of the symptoms of ciguatera or scromboid intoxication. These chemical food poisonings frequently occur within a short time after the person eats the food. The symptoms are dramatic and unusual and thus these diseases would be more likely to be detected.

Therefore, reports of those types of outbreaks, which frequently involve a small number of people may overrepresent the actual true disease effect of seafood. From 1973 to 1984 fish and shellfish accounted for 374 and 180 of the total 5,924 disease outbreaks respectively.

It is important that only about 40 percent of all outbreaks are attributed to a particular cause. So there are many people who have foodborne illness, especially of a viral etiology, where we have difficulty making a diagnosis. Those cases would not fit into any documented diagnostic category.

The CHAIRMAN. "New Scientist" magazine noted a large increase in campylobacter poisoning over the last 6 years in Britain.

Is there a similar trend here in the United States?

Dr. COHEN. We have only been identifying the campylobacter organism since the late 1970's. So this is an organism that was recently recognized.

Data on campylobacter are a very good example of some of the limitations on the use of outbreak information. Foodborne outbreaks caused by campylobacter are only a very small portion of all the foodborne outbreaks, yet sporadic campylobacter infections account for—of the 6 million estimate—probably one-third of those total cases of foodborne disease in the United States. Campylobacteriosis is a significant problem.

The CHAIRMAN. Are foodborne illnesses on the rise, generally, in the United States?

Dr. COHEN. If one looks at specific diseases like salmonellosis, there have been significant increases in the last 10 to 15 years.

If you look at the outbreak data, all the other etiologies, such as staphylococcus or Clostridium perfringens food poisoning, have had little or any change. The major cause which is contributing to this overall increase in the foodborne outbreaks is salmonella.

The CHAIRMAN. Just to point it out, so that laymen will understand, how many Americans do come down with food poisoning each year?

Dr. COHEN. We have estimated about 6.5 million.

The CHAIRMAN. Come down with food poisoning?

Dr. COHEN. Yes.

The CHAIRMAN. How many Americans die each year of food poisoning?

Dr. COHEN. The estimate has been about 9,100.

The CHAIRMAN. So we have 6 million Americans getting food poisoning and 9,100 dying of food poisoning?

Dr. COHEN. Yes.

The CHAIRMAN. It is a pretty significant health problem then.

Dr. COHEN. Yes.

The CHAIRMAN. Dr. Leighty, you said in your prepared testimony "most food animals are found to be healthy at the time that they enter the meat supply." Does that mean that most animals arrive at the processing plant healthy?

Dr. LEIGHTY. Yes. They arrive at the processing plant healthy, and they are found, when we conduct a detailed inspection of the animal, to be healthy and suitable for food.

The CHAIRMAN. The National Research Council report on poultry inspection stated that chickens transported in the same truck often had the same bacterial infections. Does that seem supportable by data you have seen?

Dr. LEIGHTY. Yes. Generally, they have originated from the same production stock, they are genetically very similar, all of the same age, all raised together in the same building. So there is always a close relationship of any communicable disease that might be found among them.

The CHAIRMAN. In your prepared testimony you gave the percentages of carcasses condemned at post mortem. The overall rates were really low, not more than 1 percent for any meat group, but even within that 1 percent it varied considerably among the various meat groups, from beef to poultry to pork.

What is it about the underlying nature of the animals that makes for these differences?

Dr. LEIGHTY. Probably the husbandry methods, since the poultry and swine are generally raised in groups. The frying chickens, there may be 4, 5,000 raised in the same house. So if they are healthy, they generally are very healthy; if they have a problem, it may affect many animals.

The same thing is essentially true of swine raised very closely. Cattle of course are fed together, generally, but the transmission of disease is not the same problem.

The CHAIRMAN. If you were able to develop all the data you wanted, even the methodology you wanted, don't you have to know what specific farm, what feed lot particular animals come from, to be able to have the best information?

For example, if you have an A, B, and C farm, or an A, B, and C feed lot, all for the same kind of animals, isn't it a fact that the health of the animals could vary?

Dr. LEIGHTY. That is right. It is important for both public health reasons, looking at it from a food hygiene point of view, as well as from an animal health point of view. While we are talking of food hygiene, the threat of foreign animal diseases makes it also important to be able to trace these animals back to their point of origin.

But the best kind of a food hygiene program is a preventative program that eliminates disease in food animals and sends healthy animals to slaughter, rather than having to inspect and condemn animals that are unfit.

The CHAIRMAN. Thank you. Senator Cochran.

Senator COCHRAN. Mr. Chairman, thank you very much.

Is it fair to say that the food in our country, in the United States, is probably the safest for the consuming public, of any country in the world?

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Dr. LEIGHTY. It might not be fair to some other countries that are doing a good job to say that, but it is fair to say that there probably are few countries that are doing a better job than we are in that respect.

Senator COCHRAN. I think it is important that we are looking into the scientific basis for food inspection, trying to determine ways to make absolutely certain, if we can, that the food the consuming public buys is safe, and fit for human consumption. I think that is a very important responsibility that the Congress has.

This oversight exercise that we are undertaking today is an effort to discharge and meet that responsibility.

Do you have any observations, from what you know of the Food Safety and Inspection Service, and its procedures, about how the inspection process could be improved from a scientific standpoint?

Dr. LEIGHTY. The National Academy of Sciences report, we have felt was a very astute report, its recommendations for the program are accepted within the program, and we believe that their many suggestions would improve our program, if they could be followed.

Senator COCHRAN. Does the other witness——

Senator PRYOR. Senator Cochran, excuse me. I am having real difficulty hearing the doctor. Thank you, Senator Cochran.

Senator COCHRAN. Could you repeat your answer for us?

Dr. LEIGHTY. Yes. The recommendations of the National Academy of Sciences committee that were made recently, as well as the original report that was made last year, we believe were very good recommendations to be followed in the future for making significant improvements in that program.

Senator COCHRAN. Would the other witness please respond, too, to the question that I asked.

Dr. COHEN. I am really not familiar. That is not my area of expertise. I would say that in the types of surveillance systems we conduct, a critical element is always evaluation of the efficacy of a surveillance system, and therefore we try to evaluate, that a system we are using is giving its desired effect, and we generally feel that is a critical element in any system.

Senator COCHRAN. Thank you very much, Mr. Chairman.

Senator MELCHER [presiding]. Senator Pryor.

Senator PRYOR. I am having one great difficulty through this whole debate on this issue. There is a debate about this whole matter of food safety, and we are right in the middle of it, and we want to do something about it, and I think we are going to do something about it.

But we find it very, very difficult to arrive at the proper facts and figures. For example, this morning, in looking over the prepared statements submitted by the witnesses prior to their oral testimony, we are seeing great variations in the causes, in the number of outbreaks, and the potential for food poisoning of some nature. And I think that we must—and I underline this, Mr. Chairman—we "must" find the right data, or I do not know how we are going to come up with the right solution.

For example, a group called Public Voice, has stated recently in a press release, that 24 percent of the food poisoning is related to seafood. I do not know whether that is right, or not.

I do not know whether that jives with what you gentlemen are saying. But we are in a "sea" here of facts and figures, and percentages, and decimal points, and it really is confusing the issue.

Let me ask a question that the housewife out there might be interested in. Let's see if we can relate this answer to that housewife.

Let's say she goes this afternoon and buys a chicken, or a steak, or some porkchops, or whatever, at the local market, and it has been inspected and there is a stamp on there that this has been inspected. The housewife believes that it is safe to eat and safe to feed her family.

Now, what chance, if that particular product is handled correctly—if it is washed, if it is cooked at the proper temperature, or whatever—what chance is there today, that housewife, or her family, is going to contract some sort of food poisoning from that product?

What would be a percentage there? Would you venture to say that?

Dr. COHEN. I would say very small.

Senator PRYOR. Very small?

Dr. COHEN. Very small.

Senator PRYOR. Less than 1 percent?

Dr. COHEN. If you look at the bacterial etiologies which are the major recognized cause of foodborne disease, proper handling of raw foods, appropriate cooking, and proper handling of cooked foods are going to make those foods safe.

The risk that an individual will have, if they use proper food handling, is small. But unfortunately, the rates of disease do not reflect that people are properly handling foods.

Senator PRYOR. Does the data that we have today indicate if, when there is an outbreak of salmonella, or any other of the poisoning that we have talked about, we can trace whether or not that outbreak was caused from home preparation or home consumption vis-a-vis restaurant or public consumption?

Do we have that data available to us? Traceability?

Dr. COHEN. Yes. It would be possible to do that. We receive data which allows us to look for what the error is that leads to the particular outbreak. In most instances, when one is identified, it is usually that the food has been held at an improper temperature, which allows bacterial replication.

Senator PRYOR. So, many times, the cause is not at the processing plant, is this correct?

Dr. COHEN. Well, it goes hand in hand. The presence of organisms plus the presence of poor food handling leads to disease. If there are no organisms, there will not be disease. If organisms are not introduced into the food, or if there is proper food handling there will not be disease.

Senator PRYOR. Do you use, in gathering information and data, something called a multiplier? For example, I think in your statement, or it may be in our other witness' statement, there was a statement that 29.5 percent of the cases go unreported.

I do not know how in the world we can establish this.

Dr. COHEN. For example, in the 1960's and 1970's, we carried out investigations of outbreaks of salmonellosis, where we went out

into the community and went door to door and asked people whether they were sick.

We then compared the results of that survey to the reports that we had received through physicians, and through various hospitals. In those outbreaks, we generally found that there were 100 ill people for every 1 that was reported.

In the very large Chicago outbreak which occurred a couple of years ago, we had a chance to go out and to do a similar type of survey, and we found that where we had some 16,000 or 17,000 reported cases—meaning that someone had isolated a salmonella from a patient and reported it to the health department. When we actually were able to question people over the telephone to determine the presence of disease, we estimated that there were almost 200,000 people who were affected.

So in this outbreak 1 out of 10 cases were reported. A reasonable estimate for the magnitude of unreporting is somewhere between 90 and 99 percent.

Senator PRYOR. To both doctors, I say thank you. I may have another question or two later. I do not want to take all the time now, and I yield back, Mr. Chairman.

Senator MELCHER. Senator Bumpers.

Senator BUMPERS. To either one of you gentlemen. These days, so much food is what we call further processed. It has been the salvation, for example, of the poultry industry, and perhaps to a lesser extent to the beef industry. And you also have businesses like Giant, which is the big grocery chain here, that have expanded their outlets into further processed foods, so that if you are on your way home from work you can buy whatever you want and throw it in the oven. It is almost the same thing. That has become very trendy, and I think that is going to develop even to a greater degree.

With this in mind do you have any statistics on the difference in the outbreaks of any of these diseases, particularly salmonellosis, from further processed foods as opposed to fresh foods?

Dr. COHEN. I do not have that data. I would be concerned about the accuracy of that data because one of the strong biases in having an outbreak reported is in association with a commercial product, something that exposes more people to illness.

Household illness from a smaller food item is not as likely to be reported as a commercially available food item that causes disease in larger numbers of individuals.

Likewise, a larger food item which might make more people ill, for example, turkey, might be more likely to be recognized as a cause of disease than, say, an individual chicken which generally feeds a smaller number of people.

So, I think that those inherent biases would affect the reliability of those data.

Senator BUMPERS. Well, does FSIS have inspectors in plants that are actually cooking beef, or poultry, or some other product, before it is packaged and frozen?

Dr. LEIGHTY. Yes. Each plant that is involved in that kind of an activity is required to be inspected. The inspection may cover the plant for a relatively short time, or the inspector may be a resident there who is there all the time, whenever a plant is in operation.

We are currently studying the ways of providing more appropriate inspection, inspection that is more appropriate to the nature of the operation, than we have in the past.

Senator BUMPERS. At what temperature is a salmonella bacteria destroyed, killed?

Dr. LEIGHTY. At what time and temperature——

Senator BUMPERS. What temperature? In other words, if you are frying a piece of chicken that has a salmonella bacteria on it some place, and you put it in the frying pan to fry it, at what temperature is the bacteria destroyed?

Dr. LEIGHTY. Oh, a rule of thumb might be—the lowest temperature would be about 145 °F. A very short dwell time at that temperature would destroy the organism.

Senator BUMPERS. Poultry, for example, is never served raw, is it? Do you know of any ways that it is prepared raw, where people would eat raw poultry?

Dr. LEIGHTY. Most people do not do that, intentionally. I think some Asians, oriental people that prepare salads and things, do utilize raw poultry, but that is uncommon.

Senator BUMPERS. Similar to sushi?

Dr. LEIGHTY. Well, it is similar in that it is raw.

Senator BUMPERS. Well, if you are in one of these further processing plants where they are taking chicken fingers, or steak fingers, or whatever, and they are cooking it, and, normally, I do not know what temperature they cook it at. But if you cook chicken fingers, or chicken patties, or whatever, and you cook them thoroughly and you freeze them, what would be the chances of getting salmonellosis from that?

Dr. LEIGHTY. If the process is properly controlled in the plant, probably near zero, unless it would be contaminated from another source somewhere along the way.

Senator BUMPERS. And of course we have no way of knowing when there are incidental cases, whether it came from the product, or something else in the kitchen, or in the dining room, or whatever, do we?

Dr. LEIGHTY. That is true.

Senator BUMPERS. And if a housewife takes a chicken, even if she does not wash it—and I think probably the high percentage of housewives who buy a fresh piece of meat at the market, they take it home and they cook it, and they usually cook it pretty thoroughly—but the first thing they do is to wash it. Wouldn't that be correct?

Dr. LEIGHTY. Ordinarily, I think that would be true.

Senator BUMPERS. But even so, if they wash it, and it still had some residue embedded in the skin—of salmonella bacteria or something—as long as they cook it, they should not get salmonellosis unless they had their hands on something else that has a bacteria on it, should they?

Dr. LEIGHTY. That is right. The outbreaks that we have seen, that have been significant commercially, have occurred where utensils, trays, things like that, have been used for handling cooked poultry after they have been used to handle fresh poultry.

Senator BUMPERS. Well, packing houses and poultry processing plants are very complicated businesses, and when you are taking a

live animal and bringing out a wholesome product at the end, that is a complicated procedure, isn't it?

Dr. LEIGHTY. Yes.

Senator BUMPERS. Well, let me just ask you a couple of other questions. Is the inspection system that you use in a processing plant, is it more designed to find a diseased animal, or a diseased carcass, or, is it more designed to pick up salmonellosis?

Dr. LEIGHTY. It is designed to pick up carcasses that exhibit gross evidence of disease, disease that is readily apparent to an inspector as the animal passes him on the line.

Senator BUMPERS. And of the roughly 55 million carcasses of chickens that are condemned each year, most of that is because of disease? They are not thrown off because of salmonella, is that correct?

Dr. LEIGHTY. That is right.

Senator BUMPERS. Now let me ask you one other question that is not really totally related to what we are here for this morning, but I am really curious about it, and I have been for many years. I used to raise cattle, and, when you had a sick calf, it was customary to give that calf a shot of combiotic. Now combiotic is called that because it is a combination of antibiotics. Is that right?

Dr. LEIGHTY. That is right.

Senator BUMPERS. Is that still prevalent?

Dr. LEIGHTY. Combiotic is still used.

Senator BUMPERS. And is there any way of detecting the level of antibiotics in the carcass of an animal at the processing plant?

Dr. LEIGHTY. Yes. Not at the processing plant. We have methods of determining whether or not an antibiotic has been used, but as yet we cannot determine which particular antibiotic was used. We use a method that is very similar to the methods that physicians use to determine the sensitivity of an organism to an antibiotic. That is, we drop a swab from the animal's tissues onto a plate that has been seeded with sensitive bacteria, and look for an area of growth inhibition.

Senator BUMPERS. Are those spot checks, or do you do that on every carcass?

Dr. LEIGHTY. It is spot checking.

Senator BUMPERS. Is it possible for people to become immune to antibiotics from eating animals that have a high level of antibiotic in them?

Dr. LEIGHTY. Is it——

Senator BUMPERS. Is it possible for me, as a human being who consumes quite a bit of beef and poultry—is it possible for me to develop an immunity to the effectiveness of antibiotics, if I eat enough beef that has a high enough level of antibiotics in it?

Dr. LEIGHTY. That is a question about which there has been a great deal of debate over many years, but——

Senator BUMPERS. I think it ought to be the subject of another hearing here, but I was just curious about it.

Dr. LEIGHTY. There is a good deal of evidence that can occur. That the organisms that might affect your body may carry resistance to antibiotics as a result of the use of antibiotics in animals.

Senator BUMPERS. Thank you, Mr. Chairman.

Senator MELCHER. Dr. Leighty and Dr. Cohen, I have been surprised this morning to learn that it is a positive relief to have witnesses in front of us that are testifying on scientific facts. Now I have been pondering in my own mind why it is such a relief, having been exposed to it for so long in college, and when I was in practice, trying to keep up on the research and developments of veterinarian medicine through reading the journals.

I found myself often bored and my mind wandering. I think the answer to my relief is that I have been exposed to so many assertions, and so much dialog, and speeches in the committee and on the Senate floor, that really are not based on fact, but are too often based on conjecture and opinion. I want to compliment you both on your testimony, but that does not mean I do not have questions for you, because I do think you leave some gaps.

First of all, Dr. Leighty, while you gave a very brief outline of what the inspection in meat and poultry involves, I would like to ask you the pertinent question. How much does this cost per pound? Let's start with poultry.

Dr. LEIGHTY. I do not have that information. I am sorry.

Senator MELCHER. Is it an infinitesimal amount?

Dr. LEIGHTY. Yes. It is a relatively small amount.

Senator MELCHER. We are talking of mils, or fractions of mils per pound?

Dr. LEIGHTY. Yes. That is true.

Senator MELCHER. And how about for beef and pork, and whatever other meat animals you inspect?

Dr. LEIGHTY. In aggregate, the cost for operating an Agency such as ours is significant. It costs millions of dollars to operate it.

Senator MELCHER. It costs about $340 million a year.

Dr. LEIGHTY. Yes. But looking at a pound of beef, or a pound of pork, a pound of chicken, it is relatively little.

Senator MELCHER. Well, do you inspect as much as 20 billion pounds of beef per year?

Dr. LEIGHTY. Yes; 33 million cattle, and we reinspect imported meat.

Senator MELCHER. Could I say that it is much less than 1 cent per pound?

Dr. LEIGHTY. I think that would be very close.

Senator MELCHER. It obviously would be much less than 1 cent per pound. What has been the trend over the past several years? Have you been cutting the number of inspectors?

Dr. LEIGHTY. The number of inspectors have been reduced, and the productivity of those that remain has been greatly increased.

Senator MELCHER. The number of inspectors has been cut?

Dr. LEIGHTY. Yes.

Senator MELCHER. And that is in total numbers?

Dr. LEIGHTY. Yes.

Senator MELCHER. And the numbers of veterinarians, as a part of that group, has been cut, is that correct?

Dr. LEIGHTY. Yes. Not to the same extent as their inspector assistants, but the number of veterinarians has been cut.

Senator MELCHER. Veterinarians, as part of the total number of inspectors, has been cut?

Dr. LEIGHTY. Yes.

Senator MELCHER. So the whole number has been cut, and out of that, the veterinarians have been cut?

Dr. LEIGHTY. Yes.

Senator MELCHER. Now why is that? Isn't that to save money?

Dr. LEIGHTY. Yes.

Senator MELCHER. Hasn't it been driven by OMB?

Dr. LEIGHTY. Yes. We must operate as economically as possible and there is continuing pressure to do that.

Senator MELCHER. Isn't there pressure to even eliminate a veterinarian making the ante mortem inspection, and leaving that to somebody who is not a veterinarian?

Dr. LEIGHTY. I do not know of any definitive effort to do that. I do not know that as a matter of policy, that has been recently proposed.

Senator MELCHER. It has been proposed?

Dr. LEIGHTY. I say I do not know that as a matter of policy, that it has. I do not believe that it has.

Senator MELCHER. Would you oppose it, if it were proposed?

Dr. LEIGHTY. I believe that there should be supervision over both ante mortem and post mortem inspection by medical professionals.

Senator MELCHER. Now if this is being driven just on the basis of cost, what we are talking about to the American consumer is that we are going to save a fraction of a cent by having a little less inspection. Isn't this a proposition that ought to be made clear to the public?

In other words, if the inspection is important, as you believe and I believe—in fact we should put it more strongly than that, absolutely essential—then isn't it also essential not to retreat on the quality of the inspection on the basis of just saving a few pennies?

Dr. LEIGHTY. That is true.

Senator MELCHER. You mentioned a series of diseases that are transmissible from animal to man, but the ones we are really zeroing in on today, as foodborne diseases, are salmonellosis and the other ones that are cited by Dr. Cohen.

Nevertheless, the cost of what you do goes much beyond just an outbreak of a bacterial foodborne disease. You mentioned toxoplasmosis, and even rabies, though that would be very difficult to transmit through food, would it not?

Dr. LEIGHTY. We do not know of any cases in which rabies has been contracted from consumption of food. There could be some hazard in handling recently infected tissues from an animal.

Senator MELCHER. Which would really be the hazard primarily of the inspectors, would it not?

Dr. LEIGHTY. Inspectors or the butchers.

Senator MELCHER. Dr. Cohen, your prepared testimony informs us that beginning in 1978, the CDC found it necessary to add another pathogen, another bacterial pathogen to the list of reportable diseases.

I find myself a little bit at a loss to even pronounce it.

Dr. COHEN. Campylobacter?

Senator MELCHER. Pardon me?

Dr. COHEN. Campylobacter?

Senator MELCHER. Yes. Where are you finding that?

Dr. COHEN. It is a good example of the differences between outbreak data and sporadic case data. The few outbreaks that have been reported over the last several years have been associated primarily with raw milk as a major vehicle for the transmission of the organism.

Senator MELCHER. Is raw milk consumption going up, or down?

Dr. COHEN. I think that those outbreaks occurred most frequently among children visiting farms as a school activity, and in similar functions. I do not have any figures on the annual consumption of raw milk, so I cannot answer that. The sporadic cases of campylobacter have been studied in several instances, and, whereas in the outbreaks, it seems that vehicles like raw milk may be important—in the sporadic cases, an epidemiologic association is found in chicken products, in between 50 to 70 percent of cases.

Thus in these sporadic cases, it appears that a prominent vehicle for transmitting the disease is chicken.

Senator MELCHER. It could be eliminated in milk by pasteurization?

Dr. COHEN. Yes.

Senator MELCHER. No question of that. Then, in chickens, you get all this information based on some public health agency reporting to you, or some doctor, on his own, reporting to you, is that not correct?

Dr. COHEN. In some instances, certainly for the outbreak data. For sporadic case studies, we and other agencies have conducted studies in which we do very intensive questioning of people who are ill, as well as people who are not ill, and compare the differences. Researchers also examine laboratory characteristics of organisms that are isolated from ill people, as well as organisms that are isolated from food products to show associations.

We have conducted a study in Georgia which gave results similar to an FDA study in the Seattle area which attributed a high proportion of campylobacter cases to chicken products.

Senator MELCHER. It is not clear in my mind yet, how many physicians are reporting through the chain on that. Now let's say some physician, right here, in the District, or in Maryland. How many physicians are diagnosing and reporting it?

Dr. COHEN. It is a voluntary system that is initiated by an individual notifying a State health department of a problem.

Senator MELCHER. All right.

Dr. COHEN. And then the State health department would investigate to determine if it was an outbreak of disease, and then do an investigation, the results of which would then be reported to CDC. It would be difficult to determine how many physicians are participating in the system.

On the other hand, for other individual diseases such as the salmonellosis, the system may be different. A physician, in most instances, will take a stool specimen, the laboratory will isolate the salmonella, and that will go to the State laboratory for further tests, and then that result is reported to CDC. There is some evidence that maybe only 50 percent of the physicians who actually culture a salmonella isolate from a patient, send it onto the State laboratory for further examination.

So, again, it is difficult to give you a specific figure.

Senator MELCHER. Are there a number of strains?

Dr. COHEN. There are over 1,500 serotypes of salmonella.

Senator MELCHER. All right. How about the other one?

Dr. COHEN. For campylobacter?

Senator MELCHER. Yes.

Dr. COHEN. That is an emerging system where we are developing serotypes, but there will be a number of strains.

Senator MELCHER. CDC has pioneered the work in typing salmonella over the past generation—I believe it is an accurate number of years, 24 years—and you have now identified 1,500 different strains. When you identify them, don't you quite often identify what region of the world they came from?

Dr. COHEN. In some instances. Also, there are predilections for certain strains with different species of animals.

Senator MELCHER. Is it true that a large number of salmonella outbreaks have been traced back to not only fish, or sea product, but also imported sea product?

Dr. COHEN. I would not say that that would account for a large proportion of the salmonella cases or outbreaks. There are surveillance systems that are specifically conducted by other agencies, that look at the various food products that are imported, and the isolates that they obtain from these studies are sent to us for serotyping, or the reports of the results of the serotyping, and these are included in our summaries.

Now if an agency has a program where they look at the specific food item, such as imported seafood, and do not look at other items, then the large percentage of the isolates attributed to this source may be a misrepresentation of the actual degree of contamination of various foods.

Senator MELCHER. In the reporting, has there been any evidence that the workers themselves in poultry plants have a higher level of incidence of salmonellosis?

Dr. COHEN. The only study that we have, where we have tried to look at occupations, would be a study we conducted in 1979 and 1984, where we looked at a number of factors associated with individuals who had salmonellosis, including occupation. I am not aware of any increased risk in that population.

Senator MELCHER. I have an economic question, Doctor. The cost of treating and of taking care of people from foodborne outbreaks is a fairly significant amount, is that correct?

Dr. COHEN. True.

Senator MELCHER. Of the reportable diseases?

Dr. COHEN. Yes.

Senator MELCHER. All right. Thank you very much.

The CHAIRMAN. Could I just interject a couple of things? I want to finish up with this panel. To follow up on something that Senator Melcher just said, I believe Dr. Archer, who will be on the next panel, published an article in 1985 estimating costs of around $2 billion in medical expenses and in lost wages, annually, for people suffering from salmonella infections. And, Dr. Leighty, I understand that in your answer to one of Senator Melcher's questions, you said that meat and poultry inspection costs a little less than 1 cent per pound.

I am told by the staff that it is about seven-tenths of 1 cent per pound, retail weight. I have something that I would hope the Department would think about. This is at a time when USDA has reduced the number of inspectors from 10,000 to around 7,000. They have done this over the past 6 years.

I think the average consumer would willingly pay that 1 cent per pound, or even a little bit more than 1 cent per pound, to know that their food is being adequately inspected. Most assume it is anyway, and most assume that that has been built into the cost. That it is costing more to wrap that food and put it on the counter than it is costing to inspect that food is a disgrace.

I would hope that USDA understands that if consumers have their choice of a colorful wrapper or an adequately inspected product, they are going to pay for the adequately inspected product. And if nothing else comes out of these hearings, I would hope that message would come out very loud and clear. I know it is certainly a message I am going to send to the Secretary, and to others. These costs are really infinitesimal, the costs of adequate inspection, as compared to the dangers we face.

Senator Pryor.

Senator PRYOR. Mr. Chairman, I will be ready in just a moment.

The CHAIRMAN. And I might mention at this point that I am going on to another hearing and would leave the Chair to Senator Melcher who, after the questions here are finished, will introduce Dr. Archer, Dr. Roberts, and Dr. Menning. Go ahead, Senator Pryor.

Senator PRYOR. Dr. Cohen, you mentioned a few moments ago in response to a question, I think, the Chicago salmonella epidemic. When was that? In 1985?

Dr. COHEN. Yes.

Senator PRYOR. Now that outbreak was caused by consuming what product?

Dr. COHEN. That was pasteurized milk.

Senator PRYOR. That was a milk issue?

Dr. COHEN. Yes.

Senator PRYOR. I did not want that to be confused with something else.

Dr. COHEN. Yes.

Senator PRYOR. And I just wanted the record to indicate that.

The CHAIRMAN. Back in my earlier statement I noted that it was the biggest salmonella outbreak that we had seen in the last several years.

Dr. COHEN. Yes.

Senator PRYOR. I think for the record, also, we have a news article published—I believe this is the Washington Post, May 22, 1987—and it is entitled "At Your Own Risk." The first paragraph says, "We're talking specifically of chickens, poultry, what we put on the table at dinner" et cetera.

Then the writer, with all good intentions, quoted a man by the name of Vernie Gee, G-e-e, a Food Safety and Inspection Service official from Long Beach, CA. Mr. Gee testified before the Governmental Affairs Committee last month, and when asked whether he had worked in a poultry plant, the answer was no. He worked in a

hog, and also in a beef processing plant. So, once again, I am trying to set the record straight as to some of these allegations.

Now we have no real inspection program for fish. The Chairman today has recommended that we go forward with an inspection program. Would you, at the Centers for Disease Control, support that proposal?

Dr. COHEN. Well, I think you have to address what diseases are being transmitted by those products, and at this point it would be worthwhile to separate fish and shellfish, because there are different epidemiologic associations.

The fish diseases we see are frequently chemical intoxications, and so to have any type of an effective system we would probably need chemical assays for the various toxins that will be producing the disease.

For shellfish, much of the disease which may be transmitted may be viral illnesses, in which it is very difficult to make a diagnosis in people, and it may be very difficult to identify the organism in the food itself.

Again, we have a problem, to some extent, with shellfish, that some people like to eat it raw, which is not going to destroy any of the agents that are able to be killed by heating.

Senator PRYOR. Well, you really did not give me an answer, but I am going to take what you said at face value. As for myself, I support at least some sort of an inspection program for fish.

The fourth issue I would like to bring up—and I think Senator Melcher brought this issue up—is the decline in the number of inspectors in our processing plants.

Now my question is, if we increased the number of inspectors in the plants, can an inspector with his, or her, naked eye see salmonella, or see any of these organisms in poultry or in beef, or in swine, or in any of the other inspected products? Can you see that with the naked eye?

Dr. COHEN. That is really out of my area of expertise. I would pass that on to the U.S. Department of Agriculture.

Senator PRYOR. Doctor, did you want to comment on that?

Dr. LEIGHTY. Yes. The answer is no, these are microscopic organisms that occur generally as contamination on animals that were healthy at the time of slaughter.

Senator PRYOR. I see. These are all the questions I have, and I want to thank both of you.

Senator MELCHER [presiding]. Dr. Leighty, I do have another question for you, and it is not an afterthought. I just waited until now because I wanted a chance for others to ask questions. Dr. Leighty, not only have there been fewer inspectors available to inspect as large a quantity of poultry and meat, but it has also been true that the inspectors, both veterinarians and lay inspectors, have been subjected to looking at more animals, more product per hour. Is that not correct?

Dr. LEIGHTY. That is true.

Senator MELCHER. I would assume that somebody has had a study on this that would try to attempt to justify it. I just want to ask you, in your own professional opinion, isn't that taking a chance?

Dr. LEIGHTY. If the data that has been produced by the people that are doing that kind of management planning is what it is purported to be, then the risk in the way that they are inspecting should be very, very low. The emphasis has been on attempting, through more scientific application of the activities of these people, to make better use of them in an attempt to do a better job with fewer people in the interest of economy.

Senator MELCHER. Well, I am not too sure what you said, Doctor, but I want to get to the root of it. Is the data good enough?

Dr. LEIGHTY. I am not really qualified to answer that question. I am not involved in that kind of study. I am a staff person that is really concerned with animal disease diagnosis, and that sort of thing.

Senator MELCHER. One final question, then. Let me state, however, before I ask this last question that I think the data on which these studies are drawn has to be professionally related, and interpreted by the people in your division, and if it has not been, it should be.

But the last question is this. While the number of inspectors has been going down, the number of animals per inspector, per hour, has been going up, is that not correct?

Dr. LEIGHTY. That is true.

Senator MELCHER. All right. Thank you both very much.

Our second panel is Dr. Douglas Archer, Director of Microbiology, Food and Drug Administration; Dr. Tanya Roberts, Agricultural Economist, Commodity Economics Division, Department of Agriculture; and Dr. Edward Menning, executive vice president of the National Association of Federal Veterinarians.

Welcome to the committee. We are not choosy on who proceeds first, but the first named on the panel is Dr. Douglas Archer.

STATEMENT OF DOUGLAS L. ARCHER, DIRECTOR OF THE DIVISION OF MICROBIOLOGY, CENTER FOR FOOD SAFETY AND APPLIED NUTRITION, FOOD AND DRUG ADMINISTRATION, U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

Dr. ARCHER. Thank you, Mr. Chairman. Mr. Chairman, committee members, I am pleased to respond to the committee's invitation to answer questions regarding the long-term effects of foodborne disease.

I am Douglas Archer, Director of the Division of Microbiology, Center for Food Safety and Applied Nutrition of the Food and Drug Administration.

In lieu of written testimony I have submitted published papers concerning my thoughts on foodborne disease, and am prepared to answer any questions concerning that issue.

Thank you.

[The articles by Dr. Archer follow:]

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Incidence and Cost of Foodborne Diarrheal Disease in the United States

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ABSTRACT

An estimated 68.7 to 275 million cases of diarrheal disease episodes from all causes occur annually in the United States, representing an average of 0.29 to 1.1 cases per person per year. The total number of cases of foodborne origin and subsequent person-to-person transfer was estimated to be at least 24 million and perhaps as many as 81 million or more cases per year. Updating previously published patient cost estimates, including lost wages as well as direct medical costs, the average estimate-based value for food-associated illness is in the billions of dollars per year. Scientifically established chronic sequellae to diarrheal disease further increase the total economic burden but cannot be estimated from available data. Other associated clinical problems that are likely to be related to acute diarrheal episodes would further increase costs.

Hauschild and Bryan *(40)* estimated the cases of foodborne and waterborne illness in the United States to be 1.4 to 3.4 million per year. The estimate was based on results of follow-up surveys conducted by the Centers for Disease Control (CDC) after various disease outbreaks. The ratio of estimated cases vs. those initially reported for all foodborne diseases was 25:1 *(40)*. Todd *(79)* estimated the number of foodborne disease cases in the U.S. to be 5 million per year.

The economic costs of foodborne disease have also been determined *(29,55)*. Most recently, Todd *(79)* estimated the cost of all foodborne disease in the U.S. to be $1 billion to $10 billion annually; this figure included such factors as direct medical costs, lost wages and productivity, and industry loss through embargo, voluntary destruction and recall.

Surveillance of foodborne disease in the U.S. is a passive, voluntary system wherein states may request the assistance of CDC. It is generally accepted that the true incidence of foodborne disease is underreported and that economic loss estimates, which are directly dependent on incidence estimates, may also be underestimated. This report estimates the incidence of total diarrheal disease in the U.S. by extrapolation from total incidence data of the National Center for Health Statistics (NCHS) and foodborne diarrheal disease incidence from the most current data available, with consideration of new pathogens and knowledge of their pathogenic mechanisms.

Previous reports, which dealt with extrapolations from foodborne incidence data only, could not present incidence estimates from the perspeciive of total diarrheal disease, and chronic conditions associated with or following diarrheal disease were not considered with regard to their impact on morbidity and mortality. Our estimate of total and foodborne diarrheal disease is the strongest argument to date for increased surveillance and research.

SCOPE OF CONCERN– SYMPTOMOLOGY AND SPREAD

Although diarrhea may be of short duration and self-limiting, it may in some instances, depending on the nature of the infecting organism or the age and health of the host, be protracted and lead to other disorders. Infection need not always present itself as diarrhea, e.g., yersiniosis may be present as severe abdominal pain without diarrhea *(77)*. Symptoms of vomiting and fever are highly variable *(12,77)* with disease of foodborne origin.

Diarrheal disease is transmitted through food and water and person-to-person (fecal-oral) contact and from living animals to man. The latter is a minor consideration in overall incidence. Much diarrheal disease previously thought to be due to person-to-person transmission is actually foodborne. It is often difficult to determine the ultimate source of an outbreak, that is, food may be the source, followed by person-to-person spread. It seems illogical to separate these two transmission modes since it is impossible in many instances to determine what proportion of total disease is caused by each. It seems more appropriate to consider that diarrhea transmitted person-to-person with foodborne infection as the principal source is a disease of foodborne origin; this is particularly true when foodborne transmission is indicated epidemiologically.

INCIDENCE OF SYMPTOMS

A "National Ambulatory Medical Care Survey" for the U.S. was conducted by NCHS for 2 years (1977-1978) *(65)*. Questionnaires completed by a cross-section of physicians nationwide yielded the following information. The number of persons per year with the given symptom as the principal reason for the visit: abdominal cramps/pain, 8,783,500; diarrhea, 2,423,500; and vomiting, 1,873,000 *(65)*. The most frequently stated second or third complaints were nausea or diarrhea. Significantly, 362,500 visits/year involved gastrointestinal bleeding as the principal reason. In summary, of the 2,423,500 principal complaints of diarrhea per year for 1977-78, 1,332,000 were diagnosed as diarrheal disease. Another 676,000 were diagnosed as diarrheal disease when abdominal cramps/pain was indicated as the principal complaint; 285,000 females with vomiting as principal symptom were also so diagnosed (data unavailable for males). An additional 454,500 diagnoses of diarrheal disease were made when principal complaints were nausea or stomach cramps, pain or spasm *(65)*. In total, approximately 2,750,000 cases of physician-diagnosed diarrheal disease occurred annually for 1977-78.

ESTIMATING TOTAL DIARRHEAL DISEASE IN THE U.S.

NCHS data indicate conservatively that 2.75 million annual diagnoses of diarrheal disease were made by physicians for 1977-1978 *(65)*. The question then becomes, How many people seek a physician's care for diarrhea or an associated symptom (e.g., abdominal cramps, vomiting, nausea)? The answer depends on many variables such as severity and duration of symptoms, which may relate to pathogen dose, the nature of the pathogen, health status/age of the host and many intangible factors such as educational level, economic status and availability and affordability of health care. For example, it was clearly shown that most children attending day-care centers were sent to the centers regardless of diarrhea *(67)*. Of the foodborne disease-associated symptoms, abdominal pain was the predominant reason that persons sought medical attention. Although severe abdominal pain plus occult blood and fecal leukocytes are strongly associated with invasive pathogens *(57)*, diarrhea does not always result. Persons with watery diarrhea only are unlikely to seek medical care unless it persists. By then, it is usually too late to obtain valid culture results. In fact, totally asymptomatic infections can occur, which can be determined only by serological or stool culture methods. Probably only one person in 25 (at best) or one in 100 (at worst) seeks medical attention. It is likely then that between 68.7 million (2.75 million diagnoses × 25) and 275 million (2.75 million diagnoses × 100) actual cases occur annually in the U.S.

This estimate is consistent with an annual rate of 224 million clinically significant enteric infections estimated in an unpublished report of the Health Policy Task Force, August 1984. (See Acknowledgments)

INCIDENCE OF FOODBORNE DISEASE

Hauschild and Bryan *(40)* calculated the number of human cases of salmonellosis per year in the U.S., using data obtained from post-outbreak questionnaires, and derived a ratio of 29.5:1 for the estimated ill to the initial human isolations; this ratio was designated R_i. The data on which this ratio was based are presented in the principal reference *(40)*. The average number of human *Salmonella* isolations/year during the period 1969 to 1978 was 25,000/year. Applying the R_i, Hauschild and Bryan *(40)* calculated an average number of estimated cases of salmonellosis for any year during the period 1969-1978 to be 740,000 cases/year (29.5 × 25,000). The number of human isolates of *Salmonella* in 1983 was 38,881 *(21)*; application of the R_i would predict that 1,147,000 human cases of salmonellosis probably occurred in 1983.

Campylobacter jejuni is a recently recognized, important cause of foodborne disease in the U.S. and abroad. A recent case-control study of salmonellosis and campylobacteriosis conducted in Seattle, WA (72) reported that the incidence of campylobacteriosis was 2.5-times greater than that of salmonellosis. Other investigators have concluded that the incidence of campylobacteriosis exceeds the combined total cases of salmonellosis plus shigellosis *(12,76)*. If the figures were extrapolated to the entire country it would mean that approximately 2,867,000 cases of campylobacteriosis occurred in the U.S. in 1983 (2.5 × 1,147,000 estimated salmonella cases). Campylobacter isolations from humans are not reportable to CDC; therefore estimates based on R_i cannot be made.

Shigella isolates from humans reported to CDC to 1983 numbered 14,946 (22). Shigellosis is thought to be transmitted primarily person-to-person; however, foodborne and waterborne outbreaks have been documented. Applying the *Salmonella* R_i of 29.5 (assuming its applicability), 440,900 cases of shigellosis would have occurred in 1983. If the estimates for diseases caused by *Salmonella*, *Campylobacter* and *Shigella* in 1983 are totalled, 4,455,000 cases of diarrhea resulted from these three pathogens.

Other well-known foodborne pathogens, such as *Staphylococcus aureus*, *Clostridium perfringens*, *Yersinia enterocolitica*, *Bacillus cereus* and *Vibrio parahaemolyticus*, accounted for 33.9, 13.4, 3.8, 0.9 and 0.2%, respectively, of confirmed foodborne disease cases in 1981 (*20*). That same year, *C. jejuni* accounted for only 5.6% of reported cases. Missing altogether were pathogenic *Escherichia coli*, *Vibrio vulnificus*, *Cryptosporidium* and enteroviruses, all of which were involved in diarrheal disease outbreaks and, in some cases, deaths in 1983, e.g., the *E. coli* strains that caused nationwide sporadic outbreaks of hemorrhagic colitis (*24,26,66*). *Y. enterocolitica*, a recognized cause of gastroenteritis and

septicemia in infants (*74*), is surely underreported. Shigellosis, giardiasis and rotavirus are leading causes of diarrhea in day-care centers. According to one recent investigation (*54*), food handling/serving plays a significant part in these outbreaks. In a prospective study of diarrhea in Houston day-care centers, Pickering et al. (*67*), determined the rate of diarrhea to be 0.34 episode/person/year for attending children and staff. Estimated attendance figures for U.S. day-centers totalled 11,000,000 children in 1984 (*23*). Based on the data of Pickering et al. (*67*), this would calculate to 3,740,000 cases of diarrhea/year among children attending day-care centers and probably another 300,000 cases of diarrhea in teachers/staff. This total far exceeds the total number of diarrheal diseases diagnosed by physicians in 1977-78, even without calculating subsequent transmission of disease to family members.

Other newly recognized pathogens may also cause foodborne diarrhea. *Aeromonas hydrophila* is only rarely associated with diarrheal disease in the U.S., yet is ubiquitous in U.S. waters (*41*). Australian investigators report that *A. hydrophila* is the leading cause of diarrhea in children (*14*), accounting for 10.8% of cases. A recent U.S. report demonstrates the tenacity often required to isolate this pathogen from feces (*64*). Enteric viruses are also fast becoming recognized as major causes of foodborne disease. Several recent outbreaks of presumed viral gastroenteritis in New York were attributed to consumption of raw clams (*17*).

The NCHS survey showed that clinical laboratory tests on patients with diarrhea are carried out in only 18% of cases (*65*), with culture procedures probably limited to *Salmonella, Shigella* and *E. coli*. The cost to the patient for specialized tests for the total spectrum of intestinal pathogens would be prohibitive; in fact, the cost/benefit of stool cultures was recently questioned (*48*). In two prospective studies relevant to the issue of stool culturing (one in the United Kingdom, the other in Switzerland), feces from persons entering hospitals with acute diarrhea were vigorously cultured and tested for most known or suspected enteric pathogens or their toxins. In the U.K. study, suspect microorganisms or toxins were found in 58% of persons with diarrhea (*44*). The Swiss study uncovered suspect causative agents in 38.7% of patients studied (*57*). Significantly, more than 30% of patients in the Swiss study with negative stool cultures had occult blood and fecal leukocytes in their stools, suggesting the existence of as yet unrecognized pathogens or pathogens not culturable under conditions employed (*57*). In both studies, *Clostridium difficile* toxins were recovered from patients with no previous history of antibiotic use. Thus two highly industrialized nations, using the best known culture methods, were only able to identify pathogens in feces about one-half the time. In the Swiss study, specific food exposure significantly correlated with *Campylobacter* and *Salmonella* infections (*57*).

ESTIMATING FOODBORNE DIARRHEA IN THE U.S.

Estimating foodborne diarrheal disease requires assumptions and extrapolations; it also requires a definition. If a person acquires diarrheal illness through food consumption and subsequently spreads the disease through person-to-person contact, logically all involved persons in the outbreak could be scored as illness due to a food source. A perfect example of the difficulties in determining the role of foodborne vs. person-to-person contact can be found again in day-care centers. A follow-up study of day-care centers in the Houston area was recently conducted by Lemp et al. (*54*); several of the authors had been involved in the original Houston study by Pickering et al. (*67*). In the original survey, *Shigella*, rotavirus and *Giardia* were the leading causative agents of diarrhea (*67*); teachers caring for ill infants and children were felt to be responsible for person-to-person transmission of disease (*67*). In the follow-up study from October 1980 to May 1981, rates of diarrhea increased somewhat, with the highest incidence rate in day-care centers where diapering by staff was combined with food preparation or service (*54*). Since only diarrhea occurring outside the centers and reported to the centers by parents was included in this study, Lemp et al. (*54*) believe that the incidence of 0.68 case per person per year is probably an underestimate. Much of what may have been considered person-to-person transmission, therefore, may be linked directly or indirectly to food; this may be particularly true for the rotavirus diarrhea in the Houston studies (*54*). In contrast, in the recently reported outbreak of Norwalk agent diarrhea in Florida, where a common food source was clearly identified, little evidence of person-to-person spread was noted (*56*).

If the foodborne rate (4,455,000 cases/year, based on *Salmonella, Campylobacter* and *Shigella*) is doubled to account for all other known pathogens transmitted by food, a trend with a historical basis, 8,910,000 foodborne diarrhea cases/year result. It would seem equally valid to again double this number, since recent history indicates that a causative agent can be found in only 1/2 of all medically investigated cases; yet evidence in the form of occult blood and fecal leukocytes indicates the presence of enteric pathogens. This second doubling factor may also account for those cases in which overt diarrhea is absent but other symptoms are present (e.g. cramps, fever, nausea). Thus approximately 18,000,000 cases/year is not an unlikely figure. One-third again as many persons may contract illness owing to person-to-person transfer subsequent to primary foodborne disease acquisition; this could raise the number of cases/year directly (or indirectly, depending on one's views) to 24,000,000 illnesses/year attributable to foodborne pathogens. This estimate assumes that the $R_t = 29.5$ (for *Salmonella* and *Shigella*) of Hauschild and Bryant (*40*) is valid. The authors point out, however, that in several large outbreaks, the ratio of estimated cases of salmonellosis to the

number of reported human isolations was 100:1. If this is assumed to be a worst-case situation, the 24,000,000 estimate (based on R_i = 29.5) may actually be as high as 81.36 million cases/year (based on a worst-case R_i of 100). In the light of all the facts concerning variables and difficulties associated with estimating true incidence, plus new epidemiologic data on virus transmission (*54,56*), this estimate does not seem unreasonable and in fact may still be conservative. Greater efforts should be made to determine the incidence of all diarrheal and foodborne diarrheal disease in the U.S. Surveillance data on infectious diseases provide public health officials with a valuable and irreplaceable source of information on which to make priority decisions; efforts must be intensified, not curtailed.

Despite vastly improved recovery techniques and the recognition of new enteric pathogens that affect large numbers of persons, the proportion of cases of unknown etiology remains constant, suggesting either that our present methods are inadequate for some causative agents or that as yet unrecognized causative agents exist. Why are funds not available for concerted efforts to determine the true incidence of foodborne diarrheal disease? The answer probably stems from the poor image of diarrheal disease held by many health professionals. Diarrhea is all too often thought of as an unpleasant nuisance, a self-limiting, acute disease with no long-lasting consequences.

CHRONIC HEALTH PROBLEMS AND OTHER RELATED COMPLICATIONS AS SEQUELLAE TO ACUTE DIARRHEAL DISEASE

Diarrhea induced by enteric pathogens in normal, healthy adults is generally an acute, self-limiting disease, although prolonged diarrhea and at times chronic and life-threatening sequellae may result. In persons debilitated by advanced age, underlying health problems, or alcoholism, enteric pathogens can nearly always be life-threatening. Children in particular are subject to numerous disorders associated with enteric infections and chronic sequellae.

TABLE 1. *Rheumatic conditions as sequellae to enteric infections.*

Condition	Target population[a]	Estimated % affected as sequellae to enteric infection	Duration of symptoms	Genetic predisposition	Reviews
Seronegative spondarthropathies					
Reactive arthritis	P/A	2-3% Sal, Cj, Shig (*47*), 2-10% Cj (*38*); 33% Ye (*47*) Eh and Gl? (*47*); asymptomatic Ye (*31, 53*); Yp (*53*)	6 mo (*47*); 22 mo (*53*); 4 to 5 yr (*63*)	HLA-B27 (*47*) other (*53*)	(*4,46 47,52*)
Reiter's syndrome (complete)	A/P	<0.1% for all three symptoms (*6*) in total population; same as for reactive arthritis (*3, 47*). Causes: Shig, Sal, Ye, Yp, Cj, Kp? (*3*)	variable with symptoms; urethritis> conjunctivitis >arthritis, >1 mo by definition (*3*) 30-50% Reiter's cases relapse for mo-yr, 15% for 30 yr (*3*)	HLA-B27 (*47*) other (*47*)	(*3,4,46, 47*)
Guillain-Barré syndrome		Very rare sequellae to Reiter's (*34*)	<1 yr usually; neurologic disorders		
Ankylosing spondylitis	P/A	0.1%-1.5% (*16,46*), Kp colonization role (*4*) Ye, Sal, Shig? (*4,16,50*)	vary from asymptomatic sacroiliitis to debilitating disease of spine, lifelong illness (*16*)	HLA-B27 (*4,16*) HLA-B27 + A2 (*16*) HLA-A28 + B27 (*16*)	(*4,16, 47,50*)
Cardiac manifestations	A	<Rate of AS, reactive arthritis; second only to coronary artery disease as cause of conduction disturbances (*8*); sequella to any seronegative arthropathy (*8*)	From onset to death (*9*) aortic regurgitation, conduction disturbances, complete heart block; frequently requires pacemaking (*8*); mitral valve damage (*70*) can be fatal	HLA-B27 (*8,9*)	(*8,9*)
Rheumatoid arthritis	P/A	Can develop from reactive arthritis (*53,63*); *Clostridium perfringens* alpha toxin? (*1*)	Life-long debililitating disease	HLA-D_w4/DR_4	(*1*)
Septic arthritis	P/A	rare: Sal, Ye, Cj	Resolves with infection	None	not available

[a]Abbreviations: P = pediatric, A = adult, *Sal* = *Salmonella* spp. *Shig* = *Shigella* spp., *Cj* = *Campylobacter jejuni*, *Ye* = *Yersinia enterocolitica*, *Yp* = *Yersinia pseudotuberculosis*, *Kp* = *Klebsiella pneumoniae*, *Eh-Entomoeba histolytica*, *Gl* = *Giardia lamblia*, *AS* = Ankylosing spondylitis, HLA = Human Leukocyte Antigen (see review in *4*).

Tables 1, 2 and 3 present some rheumatoid, nutritional and miscellaneous conditions, respectively, that are associated with or may occur after enteric infections. The amount of medical care required to treat these conditions varies greatly. In one outbreak of yersiniosis, no arthritis may be reported, whereas in another, a 4% incidence may be reported (*75,77*); this difference appears to be strain-dependent. In countries such as Belgium, where yersiniosis is endemic, particularly that caused by the O:3 serotype, the *Y. enterocolitica* serotype O:3 is thought to be the major cause of chronic joint disease (*52*). Human isolations of the O:3 serotype appear to be on the rise in parts of the U.S. (*13*). Current census and incidence figures (*16*) suggest that 250,000 to 3.5 million people in the U.S. have ankylosing spondylitis, another rheumatoid condition associated with enteric pathogens. These types of chronic diseases and their associated cost burdens accumulate in the population. Although the amount of arthropathy-associated heart disease in the U.S. has not been studied, it is significant in other countries (Table 1).

The nutritional and malabsorption conditions presented in Table 2 affect children more often than adults. The important factor is that any loss of nutritional status may greatly increase morbidity from other causes and generally erodes the overall health of the population. Particularly alarming is the apparent epidemic diarrheal disease (*23,32,54,67*), often with several pathogens in a single patient, that is affecting numerous day-care centers nationwide. Whether this is in part reponsible for the increased incidence of cryptosporidiosis (*25*) in these facilities remains to be determined.

The recently increased number of sporadic outbreaks of hemorrhagic colitis (often associated with ground beef) and the sequellae, hemolytic-uremic syndrome, has caused health officials to speculate that the *E. coli* serotypes may be the source of many of the diarrheal diseases of unknown etiology (*24*). The appearance of other adherent, enteropathogenic *E. coli* with no known associated toxins but multiple antibiotic resistance is further cause for concern (*27,51*).

THE ECONOMIC IMPACT OF DIARRHEAL DISEASE IN THE U.S.

The report by Cohen et al. (*29*) concerning patient-related costs in a salmonellosis outbreak is frequently used as the basis for more recently published cost estimates of diarrheal disease. Those estimates were based on 1976 figures and allowed $10 for a doctor's office visit and

TABLE 2. *Nutrition- and malabsorption-related sequellae to enteric infections.*

Condition	Target population	Estimated % affected as sequellae to enteric infection	Duration of symptoms	Reviews or refs
Failure to thrive with enteropathy	P[a]	2-3% of pediatric acute gastroenteritis patients (*37*)	Reversible with proper diagnosis; life-long growth retardation possible.	(*10,37,45,68*)
Failure to thrive without enteropathy	P	6% with acute gastroenteritis (*37*)	Reversible with proper long-term nutritional support; 3 wk (mean)	(*37*)
Malabsorption (mild to severe)	P/A	By definition, always occurs with G.I. tract damage	1 wk to 1 yr depending on agent and other complications	(*15,27*)
Disaccharide intolerance	P	40% with giardiasis (*82*); 64% with CNSD (*69*), 10% with acute gastroenteritis (*37*); highly variable	During acute episode to 1-5 mo, mean 3 mo (*37*); 6 mo (*71*)	
Monosaccharide intolerance	P	4-9% acute gastroenteritis admissions (*43,61*) 100% with protracted diarrhea	2-3 d as sequella to acute gastroenteritis; 21 d (mean) up to 70 d if develops during protracted diarrhea	(*61*)
Hypernatraemia	P	0.9% with acute gastroenteritis (becoming far less of a problem)	Readily reversible with proper treatment, fatal if untreated.	(*62*)
Achlorhydria	P>A	High incidence in chronic diarrhea, moderate to nil in acute (*59*)	May lead to contaminated small bowel syndrome (*36*)	
Cow's milk intolerance (non-allergenic)	P	High incidence in children with occult blood or enteropathy (*28*)	May be long-term syndrome (*80*); may cause postenteritis diarrhea	(*38,80*)
Postenteritis diarrhea	P	Dependent on presence of milk proteins in diet (*80*)	Depends on avoidance of cow's milk protein (*80*)	(*39,80*)
Intractable diarrhea syndrome	P	Treatable; starts with enteropathy; protracted; of unknown etiology (*71*)	8 d to 6 mo (*71*)	(*71*)
Steatorrhea	P>A	Probably very high incidence (*58*)	About 5 d (*45*); may lead to failure to thrive	(*45,58*)
Food allergy	P>A	Unknown; probably fairly high with any enteropathic condition	Unknown; reverses in time in some, not in others	(*33,42*)

[a]Abbreviations: P = pediatrics, A = adult, CNSD = chronic nonspecific diarrheal syndrome.

TABLE 3. *Miscellaneous conditions associated with or sequellae to enteric infections.*

Condition	Target population	Estimated persons affected with enteric infection (%)	Duration of symptoms	Reviews or refs
Hemolytic-uremic syndrome	P/A[a]	4% with shigellosis (*49*); 15% with hemorrhagic colitis (*66*). Varies with organism. Causes: Ec 0157:H7, Cj, Shig, EV, possibly any enteric pathogen (see *30*)	4-17 d hospitalization (*66*); can be fatal. On the rise in U.S. (*26*); expected to increase (*24*)	(*26,30,66*)
Hypogammaglobulinemia	P	18% with chronic-severe diarrhea (*35*)	May last 2-4 yr post-onset; contributes to high morbidity and fatal infection	(*35*)
Septicemia	P/A	Highly variable; any invasive agent; underlying health disorder increases risk	Up to 2 wk antibiotic therapy; can be fatal 38-67% (*2*)	(*2,74*)
Seizures - febrile	P	In acute cases 12-45% (*5*), 23.4% (*5*)	Short episodes preceding diarrhea	(*5*)
afebrile	P	Rare	Short episode pre-diarrhea in Cj; may recur	(*600*
Endocarditis	A	Relatively rare	Heart valve damage; permanent murmur	(*2*)
Colitis	P/A	Highly variable; almost any invasive/toxic organism; Cj, Ah pseudomembranous, Cl.d (*7*)	Transient to permanent; may not be antibiotic-related; 8-10 wk	(*44,57,64*)
Pseudoappendicitis	P/A	0-10%? (*18*)	Hospitalization up to 2 wk; unnecessary surgical procedure can lead to septicemia	(*11,18,19*)
Chronic nonspecific diarrhea	P	20-25% with acute gastroenteritis (*37*) could be Ec-related (*69*)	4-20 mos (*69*)	(*69*)
Chronic diarrhea - specific (misnomer)	P/A	16-17% with acute gastroenteritis (*37*); Ec 0111:K58:H-(*27*); Ec 0111:K58:H2(*51*)	2 wk (*27*) to 25 ± 15 d (*51*); can lead to urinary tract infection; antibiotic-resistant; recurs frequently	(*27,51*)

[a]Abbreviations: P = pediatric, A = Adult, Ec = *Escherichia coli*, Cj = *Campylobacter jejuni*, Ah = *Aeromonas hydrophila*, EV = enteric viruses, Cl.d = *Clostridium difficile*.

$139/day for hospital care. At the end of 1984, the same figures would be $30 to $35 and $400 to $410, respectively (American Hospital Association Annual Survey for 1983 and Office of Research, Health Care Financing Administration, DHHS), and income and productivity losses would be at least 40 to 50% higher. The estimated total cost to the patient of $675/person would be 2.5 to 3 times greater, or $1687 to $2025/person. Thus the total cost of salmonellosis in the U.S., based on current cost estimates and 1983 incidence figures, would be $1.9 to $2.3 billion. Cohen et al. (*29*) had factored in the cost of persons not hospitalized and did not seek a doctor's care as $125; this figure, which has also undergone inflation, was factored into the overall cost/person estimate of $675. The salmonellosis outbreak used for the calculations of Cohen et al. (*29*) was an average outbreak with variable hospitalization rates and lengths covering the entire age spectrum.

Even when children are the victims, a parent must often miss work to care for the child. The loss of productivity is not directly patient-related and is difficult to quantify. Other circumstances relative to foodborne disease costs cannot be accounted for. For example, if the general nutritional and health status of children erodes because of diarrheal disease, the increase in morbidity caused by other diseases and the resultant cost would be impossible to quantitate. The cost of disability in rheumatoid diseases, although impossible to estimate, is significant. Another set of variables includes diseases which may be associated with foodborne microorganisms. For example, *Y. enterocolitica* may trigger thyroid disease (*81*), and *E. coli* and *Klebsiella pneumoniae* may play a role in myasthenia gravis (*78*). More such associations may be found and eventually proven. These factors will undoubtedly impact greatly on total cost estimates.

Diarrheal disease from foodborne sources is generally preventable. Any money spent on research, surveillance and public education would be only a small fraction of the cost otherwise borne by the economy when disease occurs. This was recently demonstrated dramatically in a report (*73*) on a foodborne typhoid fever outbreak which showed that the costs incurred by five patients offset the cost of detection and prevention. In most instances detailed by Todd (*79*) in 1983, food supplier

losses in the form of recalls, lost business, legal fees, legal settlements and wages exceeded the medical costs and lost earnings of the victims; thus the indirect cost to the economy may equal or even double the patient-related cost estimates. Estimating the true cost of diarrheal illness and all associated economic costs is a complex task that will be dealt with at a later date.

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Enteric Microorganisms in Rheumatoid Diseases: Causative Agents and Possible Mechanisms

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ABSTRACT

The role of foodborne enteric pathogens in the development of three seronegative spondarthropathies (ankylosing spondylitis, Reiter's disease and reactive arthritis) is discussed. Although the prevalence of the HLA-B27 antigen in blood-related individuals suggests a genetic predisposition to these diseases, exogenous environmental factors are also indicated. A clinical profile is given to clarify certain relationships of the seronegative arthropathies. Evidence of the involvement of enteric pathogens in the onset of these conditions following gastrointestinal illness is considered along with the interactions of general and molecular mechanisms of the disease processes and the immune response.

In addition to immediate gastrointestinal symptoms, several common species of foodborne and waterborne pathogens may cause such debilitating diseases as reactive arthritis, Reiter's syndrome and ankylosing spondylitis. Certain individuals are more likely than others to be affected. The HLA-B27 histocompatibility antigen, possibly in combination with some other alleles, contributes to a genetic predisposition. Mechanisms by which enteric pathogens elicit such diseases could include cross-reactivity of bacterial antigens and human cell surface (self) antigens, circulating immune complexes (CICs), hyper-responsiveness to mucosal stimulation by antigen, hyper-responsiveness of lymphoid cells involved in inflammatory responses, or deposition of bacterial debris in joint tissue. There is considerable evidence that ankylosing spondylitis is caused by permanent genetic alteration of human tissue due to incorporation of bacterial genetic material via a plasmid.

Long-term effects of diarrheal episodes and diarrheal disease have been considered previously *(5)*. The present article expands the discussion of autoimmune diseases, emphasizing three arthritic conditions (ankylosing spondylitis, Reiter's disease and reactive arthritis) that are frequent sequellae to gastrointestinal infections with gram-negative enteric bacteria. Clinical evidence supporting the role of enteric pathogens in the etiology of these conditions is reviewed, as are predisposing factors and possible mechanisms.

Although the interrelatedness of the seronegative spondarthritides *(47)* or seronegative spondarthritis diseases *(78)* is still a topic of debate, the most recent rheumatology review *(78)* lists the following nine conditions as belonging to this group: ankylosing spondylitis, Reiter's disease, reactive arthritis, acute anterior uveitis, ulcerative colitis, psoriatic arthritis, juvenile chronic arthritis, Crohn's disease and Whipple's disease. The diseases are considered related because they appear to be linked to ankylosing spondylitis (AS), a rheumatoid inflammation of the vertebrae.

The major criteria which differentiate the seronegative arthropathies (arthropathy = any joint disease) from seropositive conditions such as rheumatoid arthritis are *(a)* absence of rheumatoid factors and antinuclear antibody (thus seronegative); *(b)* absence of subcutaneous (or rheumatoid) nodules; *(c)* inflammatory peripheral arthritis usually asymmetrical; *(d)* radiological evidence of sacroiliitis, with or without AS; *(e)* overlap of symptoms between members of the spondarthritis group; *(f)* familial aggregation or increased prevalence of disease in blood relatives; and *(g)* a prevalence of the HLA-B27 antigen in persons with the diseases. While familial aggregation is generally evidence for involvement of genetic or environmental factors, the fact that monozygous twins may be discordant for AS strongly suggests that exogenous environmental factors play a role.

HUMAN LEUKOCYTE ANTIGEN (HLA)

The HLA system [recently reviewed by Carpenter *(11)*] was identified as an antigen group separate from the erythrocyte blood group antigens when it was discovered that multiple-transfused individuals, transplant recipients and multiparous females developed antileukocyte antibodies, which react with certain white blood cells in agglutination and cytotoxicity assays. Grouping a large collection of such antisera according to reaction patterns suggested that alleles (alternative genes) were being detected. The HLA antigens are coded for on at least four or five loci (A, B, C, D and DR) on the short arm of chromosome 6 and within the major histocompatibility complex genes. An offspring inherits one A allele from one parent and most likely a different one from the other

parent. The same holds true for the B, C and D (and DR) loci. The alleles of the HLA system code for antigens which are present on most body tissues, including B- and T-lymphocytes and platelets, but not red blood cells, in varying amounts. The major antigens are subdivided into two classes. In both class I and class II, the antigenic material is glycoprotein. Class I antigens are coded by the HLA–A, –B and –C loci; presently there are more than 60 defined A and B antigens and 8 C antigens. Class II antigens are coded by the more recently recognized HLA–D locus and are defined by use of one-way mixed lymphocyte responses. Serologically, in contrast to HLA–A, –B or –C antigens, the defined antigenic specificities of HLA–D are not found on platelets or unstimulated T-lymphocytes and are coded by closely linked genes called HLA–DR (D-related). The HLA–DR system is becoming extremely important with regard to disease associations.

CLINICAL PROFILE

A clinical profile of the seronegative arthropathies is considered here with regard to those environmental triggers, the enteric pathogens; a general background discussion, therefore, is required to clarify certain disease relationships.

Either reactive arthritis or Reiter's disease may occur in a certain (variable) portion of the affected population. A percentage of those individuals who develop reactive arthritis or Reiter's disease as a sequella to gastroenteritis ultimately show signs of AS or permanent damage in joints other than the spine. Sometimes this is expressed as classical (seropositive) rheumatoid arthritis.

Reiter's disease may be more properly termed Reiter's syndrome, as it is a triad of symptoms, one of which is an aseptic arthritis. In a recent complete review and discussion, Keat *(34)* proposed that reactive arthritis is incomplete Reiter's syndrome, i.e., it is identical to the aseptic arthritis which is one part of the Reiter's triad. The concept of "incomplete" or "atypical" Reiter's syndrome was also discussed by Ford *(22)*.

Reiter's syndrome of the classical sort is characterized by post-diarrheal onset of urethritis, conjunctivitis and migratory polyarthritis. Keratotic skin lesions may occur, although they are not always part of the classical triad. Initiating infections that elicit the onset of the triad are either genital *(Chlamydia trachomatis, Ureaplasma urealyticum, Neisseria gonorrhea)* or enteric *(Shigella dysenteriae* or *Shigella flexneri, Salmonella* spp., *Yersinia enterocolitica, Campylobacter jejuni* and possibly others). Predisposition to arthritic symptoms following genital or enteric infection is associated with the HLA-B27 antigen. About 8% of the Caucasian population in the United States is HLA-B27-positive *(21)*, whereas 60-80% of patients with Reiter's syndrome or reactive arthritis express the HLA-B27 antigen *(34)*. Thus, although HLA-B27 individuals are in the highest risk group, those lacking this antigen may also acquire arthritis following enteric infection. Reactive arthritis is an aseptic arthritis, i.e., no organisms are isolated from the synovial fluid.

Early after onset of disease, the pathology of reactive arthritis differs from that of rheumatoid arthritis, showing little evidence of synovial lining cell hyperplasia. Later in the disease process the two forms of arthritis are nearly indistinguishable pathologically *(34)*. Our picture of the immunology of the joints, however, is incomplete and at times contradictory. Jennette et al. *(32)* reported on three patients with seronegative spondarthropathy, two with AS and one with incomplete Reiter's disease. All three patients were found to have IgA nephropathy, a likely indication that CICs were being deposited. The authors speculate that HLA-B27-positive individuals may hyperreact to mucosal stimulation by environmental antigens and that this results in IgA-containing CIC. Antibody to *Salmonella typhimurium* H antigen was recovered from the sterile joint fluid of one of two brothers suffering from post-*Salmonella* reactive arthritis *(57)*. Granfors *(29)* used ELISA to detect *Y. enterocolitica*-specific IgM, IgG and IgA in studies on 356 patients whose sera gave positive tube agglutination titers to *Y. enterocolitica* O:3 and O:9 or *Yersinia pseudotuberculosis* IA. IgM antibody persisted for 1 to 3 months: IgA and IgG antibody persistence was variable. In three patients who subsequently developed reactive arthritis, yersinia-specific IgA persisted from 9 to 14 months in contrast to nonarthritic patients whose IgA disappeared within 3 months of disease onset *(29)*. Trull et al. *(71)* and Panayi *(52)* examined the sera of AS patients with either active or inactive disease (as judged by standard rheumatologic criteria) and found that IgA specifically reactive to *Klebsiella pneumoniae* was detected only in patients with active disease. Manicourt and Orloff *(45)* followed the clinical course of a patient with *S. typhimurium* gastroenteritis who subsequently developed reactive arthritis. Immune complex levels were always higher in joint fluid than in blood serum, and levels of CIC paralleled the progression of the arthritis. CICs were shown to activate both the classical and alternative complement pathways *(45)*. Theofilopoulos et al. *(69)* demonstrated the presence of C'3 and IgG Fc receptors on synovial cells. Furthermore, gram-negative bacteria that fixed complement also bound linearly to synovial tissues *(69)*. Erythrocyte-antibody-complement complexes failed to bind to synovial cells, suggesting some specificity for gram-negative bacteria. It seems possible then that antigens from gram-negative bacteria, like whole organisms, may similarly fix complement and bind to synovial tissue, thus forming the foci for inflammation in joint tissues.

The duration of symptoms also differentiates reactive arthritis from rheumatoid arthritis. Although the first episode of reactive arthritis after enteric infection usually resolves completely within 6 months of onset *(34)*, symptoms may recur. Depending on the triggering organism, 5-18% patients may have symptoms that last more than 1 year, and 15-48% may experience multiple episodes of arthritis *(34)*. In one epidemic of Reiter's disease following a shipboard outbreak of shigellosis, 9 of the 602 patients with dysentery developed the Reiter's triad and one patient was permanently disabled. Leirisalo

et al. *(41)* analyzed 160 patients with Reiter's disease and noted that those with the HLA-B27 antigen had more severely acute symptoms and developed chronic conditions more frequently. A similar observation was made by Eastmond *(17)*. Catteral *(12)* states that the relapse rate of Reiter's syndrome is 60% and that permanent joint erosion is not uncommon. In a follow-up study of 52 yersiniosis patients, conducted 4-6 years after an outbreak, Kalliomaki and Leino *(33)* found two patients with sacroiliitis and one with confirmed rheumatoid arthritis. A follow-up study of 38 (31 later confirmed) cases of suspected yersinia arthritis, conducted 4-5 years after initial diagnosis, revealed three patients with AS and one with seropositive rheumatoid arthritis. Only 6 of the 31 confirmed cases were free of joint symptoms *(66)*.

Although most reactive arthritis episodes may be of short duration, some cases may evolve into long-term debilitating disease. As Marsal et al. *(46)* noted: "Although the acute symptoms of yersinia arthritis disappear within 12 months, the long-term prognosis may be less favorable than previously thought." This observation is extendable to reactive arthritis triggered by other organisms as well.

ENTERIC ORGANISM INVOLVEMENT IN PATHOGENESIS

The clinical reports concerning gastroenteritis leading to Reiter's disease or reactive arthritis can be separated into two categories: *(a)* prospective epidemiologic follow-up studies on confirmed outbreaks of gastroenteritis, and *(b)* retrospective studies whereby arthritis is present and evidence of prior enteric infection is either lacking (i.e., the pathogen is not isolated from feces because of resolution of infection) or is obtained by serologic or other immunologic means. Obviously the former method carries more weight than the latter. No evidence should be discounted, however, as there is substantial evidence that diarrhea may not always be present in enteric infections.

The enteric organisms reported to be, or suspected of being, the causative agents of reactive arthritis, Reiter's syndrome and/or AS are listed in Table 1. The publications cited there, particularly the foreign journals, are by no means a complete reference list.

TABLE 1. *Organisms associated with seronegative spondarthropathies and selected clinical references to outbreaks.*

Organism	References
Yersinia enterocolitica	*1,3,7,8,14 15,18,22,23,29 33-37,39-41,43, 44,48,53,60,63 72,77,79*
Yersinia pseudotuberculosis	*9,13,16,39,44, 48,79*
Samlonella spp.	*17,18,34,41,42,45,56,57, 61-63,75*
Campylobacter jejuni	*7,18,34,61*
Shigella dysenteriae/flexneri	*18,22,28,34,38,49,50,65, 70*
Escherichia coli	*40*
Klebsiella pneumoniae	*20,30,52,53,70,71,73*

Historically, most reports of reactive arthritis or Reiter's disease following gastroenteritis involved outbreaks of shigellosis *(34)*. Table 1 indicates that most of the more recent reports involved arthritis (or Reiter's triad) after infection with *Y. enterocolitica* or, to a lesser extent, *Y. pseudotuberculosis*. The greatest problem appears to be monoarthritis or polyarthritis after yersinia infection, and the geographic region most often affected is Europe, particularly Scandinavia and Finland. The predominant serotypes of *Y. enterocolitica* are O:3 and O:9 *(15,37,39,40)*; however, other serotypes, such as O:4, O:7 and O:21, have also been implicated *(77)*. Although the O:3 and O:9 serotypes of *Y. enterocolitica* are more frequently encountered in Europe than in the United States or Canada, isolations in both these North American countries have been reported. In northern California, an outbreak of yersiniosis resulted in several cases of reactive arthritis or incomplete Reiter's disease *(77)*. The authors suggest that *Yersinia* may be a common cause of these illnesses in the United States *(77)*; the same conclusion has been reached by Canadian investigators concerning the cause of arthritic conditions in their country *(23)*. Curiously, *Yersinia* infection may occur without diarrhea *(15,34)* and enter a carrier state, ultimately resulting in chronic connective tissue damage *(1,37)*. Although arthritis is a frequent sequella to yersiniosis, the Reiter's triad does not occur as frequently as after infection with other enteric pathogens such as *Shigella (1)*. Investigators in the United Kingdom have suggested that even nonpathogenic *Y. enterocolitica* may play a role in seronegative arthropathies, particularly AS *(53)*.

As previously mentioned, reactive arthritis or Reiter's triad following dysentery has historically been the mainstay of the association of enteric pathogens with seronegative arthropathies. Gastroenteritis caused by *Salmonella* is also followed frequently by arthritis. Fewer reports are available on arthritis caused by *C. jejuni* and *Escherichia coli* (Table 1). Notably few, if any, reports associate *K. pneumoniae* with reactive arthritis or Reiter's disease. *K pneumoniae* is nearly always associated with AS, which is thought by some to be the end point illness of the seronegative arthropathies.

GENETIC PREDISPOSITION

The high association of persons with HLA-B27 and AS has been known since 1973 *(54)*. Ethnic grouping is also a consideration: 90-95% of Caucasians with AS have the HLA-B27 antigen, whereas only 50% of blacks with the disease have this antigen *(54)*. The association of HLA-B27 with reactive arthritis is somewhat weaker: from 72 to 84% of patients possess the antigen. Thus the association is not absolute and human genotypes other than B27 are susceptible to the seronegative arthropathies. From 6 to 8% of the U.S. population possess the HLA-B27 allele; the rate in Finland is 14%. It is not surprising on

this basis alone that Finland has a higher case incidence of seronegative arthropathies following enteric infections. One group of Finnish investigators reported that in addition to B27, a second gene (possibly C1 or C2) may predispose for Reiter's disease and reactive arthritis *(41)*. The A2 gene also has a strong association with AS, but not with Reiter's disease *(31)*. For seropositive arthropathies such as rheumatoid arthritis, still other genes (DR4 and possibly DR3) have been implicated. In two studies of yersinia arthritis, one in Finland *(41)* and one in Belgium *(14)*, opposite conclusions were reached with regard to the role of B27. The Finland study suggested that B27 may increase the severity of arthritis and be somehow involved in late sequellae such as AS, but the Belgian study noted no differences in severity of symptoms between B27 positives and negatives.

The genetics and, even more so, the genetic predispositions involved in the seronegative arthropathies are not completely characterized or fully understood. It is clear, however, that compared with a B27-negative individual, one who is B27-positive has an 18-times greater relative risk of acquiring reactive arthritis *(11)* after enteric infection, a 37-fold higher relative risk of acquiring Reiter's disease *(11,54)* and an 87- *(11)* to 126- *(54)* fold higher relative risk of acquiring AS. Whether this trend indicates a progression to AS remains to be determined. It should be pointed out again, however, that HLA-B27-negative individuals can and do acquire seronegative spondarthropathies after enteric infections.

MECHANISM INVOLVED

The underlying mechanisms contributing to acquisition of seronegative spondarthropathies after enteric pathogen-mediated gastroenteritis remain speculative. Yet, some clear trends have surfaced. For convenience, the general mechanisms involved in reactive arthritis and Reiter's disease (and possibly AS) will be treated separately from the molecular mechanisms which have been proposed for AS alone. It is curious that far more basic molecular genetic work has been conducted on AS.

General mechanisms

It has long been known that some strains of *Klebsiella*, usually *K. pneumoniae*, cross-react with the HLA-B27 antigen. There are many other such known cross reactions between various bacteria or parasites and human tissue. The underlying hypothesis for such evolutionary events is simple: It is advantageous for the invading organism to mimic the host's tissue as closely as possible so that an effective immune response cannot be mounted *(54)*. When autoimmune disease occurs, the antigenic mimicry is not perfect, but only partial. The "cross-tolerance" hypothesis of Ebringer *(19)* clearly expresses this theory wherein the patient's own antibacterial antibody attacks the tissues containing self-antigens. However, this explanation raises questions because the HLA-B27 antigen is found in varying amounts on almost all tissues except erythrocytes *(4)*. Why then does joint tissue, particularly that of the spine, become damaged by *Klebsiella*-induced autoantibody while other HLA-B27-carrying tissue seemingly remains undamaged? This question aside, there is ample evidence that antibody to the invading organism is developed and that some correlation with the clinical status exists. A recent report by Tenner et al. *(68)* expands on the observation of Theofilopoulos et al. *(69)* regarding the binding of gram-negative organisms which fix complement to synovial cells and the possibility that surface antigens shed from gram-negative organisms may do the same. Several strains of *E. coli*, including clinical isolates, bound and activated C1, the first component of the classical complement pathway *(68)*. The activation of C1 was regulated by physiological concentrations of natural C1 inhibitor *(68)*. Whether certain body sites are deficient in C1 inhibitor is not certain.

The relationship of the occurrence of *Klebsiella* in the fecal contents with AS is somewhat controversial. Although Ebringer et al. *(21)* correlated the fecal incidence of *Klebsiella* with the relative disease activity of AS, this was not supported by Warren and Brewerton *(74)*. Ebringer *(19)* did show, however, that the relationship between the fecal incidence of *Klebsiella* extended to acute anterior uveitis (one of the Reiter's triad of symptoms) as well as AS. Radiobinding assays showed that antisera to *Klebsiella* binds to vitreous humor and uveal antigens, but whether uveal antigens cross-react with (or contain) HLA-B27 was not determined *(19)*.

Crude extracts from *K. pneumoniae* induce arthritis in rats, a property shared by many microorganisms, both gram-positive and gram-negative *(18)*. The rat adjuvant arthritis model demonstrates many of the clinical features of Reiter's disease and AS, such as arthritis, urethritis, uveitis/conjunctivitis, keratotic skin lesions and ultimately, spondylitis *(18)*, and thus may be a valid model for studying the human condition.

Panayi *(52)* and Trull et al. *(70)* reported the elevation of serum IgA for *Klebsiella* in AS patients with active disease or with demonstrable *Klebsiella* in their feces. No elevation of serum IgA specific for *E. coli* or *Candida albicans* was shown. Trull et al. *(71)* extended these studies and showed a significantly higher mean serum IgA level in active AS patients than in a matched, healthy HLA-B27 control group. Again, only specific elevation of IgA antibody to *Klebsiella* was demonstrated, with no significant differences among patients and controls regarding IgA against *S. typhimurium*, *Y. enterocolitica* or *Pseudomonas aeruginosa*. Measuring in vitro antibody production, Vuento et al. *(72)* showed that pokeweed mitogen-stimulated IgA secretion by lymphocytes was identical from AS patients, persons who once had yersinia arthritis, recovered yersiniosis patients who failed to develop arthritis and healthy controls. Lymphocytes from AS patients and former yersiniosis patients without arthritis showed a higher number of IgG plaque-forming cells in response to *Yersinia* antigen. This suggests altered immunoregulation in patients with yersinia arthritis *(72)*. As previously mentioned, CICs are

often associated with enteric infections of such organisms as *Yersinia (35)* or in patients with seronegative spondarthropathies *(32)*. In the latter, IgA-containing CIC resulted in IgA nephropathy. A unifying hypothesis states that a genetically controlled hypermucosal response occurs in some HLA-B27 individuals *(32)*. It has also been suggested that the so-called enterobacterial-common antigen may play a role in eliciting reactive arthritis caused by various enteric pathogens *(2,40)*. Other investigators indicate that an exaggerated inflammatory response occurs in HLA-B27 individuals, as suggested by the presence of several factors or cell types normally associated with inflammation of the joints *(8,27,30,58,59)*.

Molecular mechanisms

Probably owing to the reported link between the fecal incidence of *Klebsiella (20,21,52,70)* and AS, although discounted by some *(73,74)*, Australian workers in particular turned their attention to possible interactions of *Klebsiella* and HLA-B27. The original experiments conducted by Seager et al. *(64)* showed two potential links: *(a)* lymphocytes from B27-positive AS patients responded poorly in vitro to *Klebsiella* antigens compared with those of disease-free B27 positives or B27-negative controls, whereas lymphocytes from the three groups responded equally to the nonspecific T-lymphocyte mitogen phytohaemagglutinin; *(b)* antibody raised in rabbits against one particular strain of *Klebsiella* lysed B27-positive lymphocytes of AS patients, but not those of B27-positive or B27-negative healthy individuals or B27-negative individuals with AS. Likewise, sera raised against other enteric pathogens were unable to lyse the lymphocytes of B27-positive AS patients. The cytotoxic antibodies could be absorbed only by lymphocytes of B27-positive AS patients *(64)*. Independently and nearly concurrently, Welsh et al. *(76)* showed that rabbits immunized with B27-positive lymphocytes produced antisera which cross-reacted with *Klebsiella* antigens and extracts from some *Y. enterocolitica* and *Shigella sonnei* (demonstrated by four immunoassays), and that antisera to *Klebsiella* lysed B27-positive, AS-positive lymphocytes. The same group further showed that human tissue typing sera for HLA-B27 had greater binding activity for *Klebsiella* extracts (three assay systems used) than non-B27 typing sera *(6)*. The Australian workers, Geczy et al. *(24)*, then showed that culture supernatant fluids of particular strains of *Klebsiella* modified B27-positive lymphocytes from healthy individuals, rendering them susceptible to lysis by antisera to *Klebsiella*, which previously *(64)* lysed only lymphocytes of B27-positive AS patients but not those of B27-positive healthy donors. Sera against other enteric bacteria failed to lyse the lymphocytes modified by filtrates of *Klebsiella*. Geczy and Yap *(26)* surveyed clinical isolates of *K. pneumoniae* and found that about 8% cross-reacted with the HLA-B27-associated cell surface marker on the lymphocytes of AS patients. Alexander et al. *(4)* studied distribution of the anti-*Klebsiella*-sensitive B27-linked component on various cells of AS patients. The component was not detected on sperm, although these cells contained the B27 antigen, but was detected on platelets and fibroblasts and on Epstein-Barr virus (EBV)-transformed lymphoblastoid cell lines derived from AS patients. The "modified" B27 was still detectable after 20 generations on EBV-transformed cell lines and after 10 generations on the fibroblasts *(4)*. These findings suggest that the modification of the B27 antigen may be genetically determined and does not require the continued presence of the *Klebsiella* factor. Sullivan et al. *(67)* characterized the *Klebsiella*-derived modifying factor and found that it was located in the outer membrane of some *Klebsiella* cells and was being excreted into the culture fluid; this cell component and excretory product was probably a glycoprotein with an acidic isoelectric point and a molecular weight of 26,000-30,000.

Prendergast et al. *(55)* surveyed 185 clinical isolates of *Salmonella, Shigella, E. coli,* and *Campylobacter* for their ability to absorb antibody activity to the modified B27 surface component on lymphocytes of AS patients. Three *Salmonella,* two *Shigella,* one *E. coli* and one *Campylobacter* species were able to do so, and thus cross-reacted with B27-positive lymphocytes of AS patients. The cross-reactive organisms elaborated a factor with properties identical to the *Klebsiella*-derived modifying factor, suggesting possible plasmid involvement in the interspecies transfer of the ability to elaborate this factor *(55)*. Cameron et al. *(10)* were subsequently able to isolate a plasmid capable of coding for the "modifying factor." Curing *Klebsiella* of the plasmid resulted in loss of its ability to elaborate modifying factors; conversely, the plasmid could be transferred to a plasmid-free laboratory strain of *E. coli,* which then acquired the ability to elaborate a modifying factor *(10)*. The plasmid's size was determined to be approximately 1.86×10^7 D *(10)*.

The problem now faced by the Australian investigators was to reconcile in vitro results with the in vivo situation. An observation of Orban et al. *(51)* aided in forming a working hypothesis. EBV-transformed cell lines from B27-positive AS patients produced a modifying factor nearly identical, both chemically and serologically, to that produced by some *Klebsiella* species and other specific-plasmid-bearing enteric organisms *(51)*. EBV-transformed lymphocytes of B27-positive healthy donors failed to produce the modifying factor. The trait of modifying factor production was stable for more than 20 generations, thus suggesting a stable genetic alteration. Although bacterially derived modifying factor could modify B27-positive lymphocytes to a form similar to B27 lymphocytes from AS patients, the factor had to remain present; when it was removed, modified lymphocytes again became resistant to lysis by antisera previously capable of lysing them *(25)* in the presence of the factor. If one assumes it to be possible that the bacterial plasmid has become stably incorporated into the B27-positive mammalian host cell, with subsequent constant production of modifying factor in vivo, the conceptual gap narrows *(25,66)*. Although the incredible similarity between bacterially derived modifying factor and that produced by AS

patient-derived lymphoid cell lines is established *(51)*, this is only circumstantial evidence that the transfer of prokaryotic DNA to an eukaryotic cell has occurred. With the plasmid in hand, the Australian workers undoubtedly are proceeding to gather direct proof of the presence of a gene that codes for the modifying factor and is identical to the plasmid gene in HLA-B27-positive lymphocytes from AS patients.

Significantly, these workers have also shown that antisera to *Klebsiella*, capable of lysing 80% of the lymphocytes of B27-positive AS patients, are cytotoxic to the lymphocytes from 60% of patients with B27-positive Reiter's syndrome and asymmetrical arthritis, and for 20% of lymphocytes from patients with B27-positive uveitis in the absence of clinical arthritis or spondylitis *(35)*. Thus these diseases may be linked mechanistically.

The possibility of a bacterial plasmid being incorporated stably into human DNA and mediating chronic disease is certainly a novel pathogenic mechanism. If proven to be correct, there will emerge an entire new field of investigation into the causes of all diseases of unknown etiology and of known association with the HLA system.

SUMMARY AND FUTURE PROGNOSIS

The ability of enteric pathogens to cause debilitating disease in the form of the seronegative spondarthropathies is well-supported by the clinical literature. The extent to which arthritic conditions in the United States are sequellae to enteric infections and gastroenteritis is not known, just as the true extent of foodborne pathogen-mediated diarrheal disease is unknown. Several questions do emerge, however, one of which relates to the frequency of reactive arthritis, Reiter's syndrome and AS in the United States versus the rest of the world. Although cases of these illnesses attributed to enteric infections are reported in the United States, most are in Europe, particularly Scandinavia. This may in part be accounted for by genetic predisposition, such as the higher percentage of HLA-B27 individuals, coupled with another as yet unknown allele which increases the chances for illness. It may, however, reflect geographic variation in the global distribution of the serotypes and biotypes of microorganisms. For example, *Y. enterocolitica* O:3 and O:9 frequently lead to reactive arthritis in Europe; in fact, in Belgium where it is endemic, these bacterial serotypes are felt to be the leading cause of acute and chronic joint disease *(37)*. Although the *Y. enterocolitica* O:3 and O:9 serotypes are infrequently detected in the United States, are they different from the European strains of the same serotype with regard to induction of arthritis? Do all bacteria that cause reactive arthritis or Reiter's syndrome harbor a plasmid(s) similar to that reported to cause AS? Would EBV-transformed lymphocytes of HLA-B27-positive reactive arthritis or Reiter's syndrome stably produce the B27 modifying factor as do EBV-transformed lymphocytes of B27-positive AS patients, or are the former two the results of bacterially produced modifying factor elaborated during gut colonization and the latter the result of true genetic alteration? Lastly, can genetically inbred animal models be developed to parallel the disease associations discussed in this review, and can they be used in research of other diseases that may be the sequellae to enteric infections? These are only a few of the unanswered questions, but the research possibilities should keep investigators busy in this area for years to come.

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Senator MELCHER. All right. Dr. Roberts.

STATEMENT OF TANYA ROBERTS, AGRICULTURAL ECONOMICS, ECONOMIC RESEARCH SERVICE, U.S. DEPARTMENT OF AGRICULTURE

Dr. ROBERTS. I am Tanya Roberts, an agricultural economist with the Economic Research Service in USDA, and it is my pleasure to have this opportunity to talk about costs of certain foodborne diseases.

It is important to emphasize that it is difficult to estimate costs associated with particular diseases, and usually requires the use of subjective assumptions. Also, researchers differ on the appropriate conceptual cost model to use. Some argue that the correct method is to add up all the direct and indirect costs to individuals, businesses, and public agencies resulting from the incidence of disease. And this is the model I am going to be using today, but I am going to focus on just two cost components that relate to individuals—medical costs and lost productivity.

Another research method, which is equally valid, is to determine the true cost by looking at how much individuals and society are willing to pay to prevent various levels of disease incidence.

In addition to the differences in methods that economists have for estimating costs of foodborne disease, the incidence of foodborne disease, as we have heard earlier, is very difficult to measure because mild cases tend not to be reported or accurately identified, and for these and other reasons, the costs that I am going to talk about today should not be taken down to the last decimal point. This is a continuing field of research, and we are developing new methodologies, new estimates, all the time.

I will talk a little bit more about the procedures. There are two types of information which are critical to estimating costs associated with foodborne illnesses: first, estimates of the number and severity of cases which occur in a population in a typical year; and second, estimates of the average medical and lost productivity costs associated with individual cases of various severity levels. Since symptoms of foodborne disease range from minor discomfort to death, it is difficult to determine the total number of cases. Epidemiological case studies have shown that incidences of foodborne disease are greatly underreported because many cases are either not correctly diagnosed, or are mild enough to not require professional medical care.

The work I am talking about today is based on a study that was published in 1986, "The Economic Losses Due to Selected Foodborne Diseases," and in that I defined three disease severity levels: mild, moderate, and deadly. Mild cases represent a day or two of illness, and are largely unreported. The number of mild cases, therefore, is inferred from the reported cases and expert opinion. Moderate cases are defined as those instances where the patient is ill for 1 or 2 weeks and seeks professional medical care. These are primarily the reported cases to CDC.

In some circumstances, foodborne disease can result in death. Although frequently reported, some deaths that result from foodborne disease are incorrectly attributed to other causes. Thus, cost

estimates must include reported deaths as well as an estimate of incorrectly diagnosed or unreported deaths.

In the cases where deaths occurs, there are a number of procedures that economists have for estimating the value of life and I am using one for the low estimate and another for the high estimate.

The low estimate is based on work published by Dolan, Hodgson and Wun which estimates the individual's contribution to future production, discounted to the year of death. The second measure is from Landefeld and Seskin, and includes values for nonlabor income, risk aversion, and a higher valuation on future earnings than the first measure. It should be noted that both of these estimates provide dollar amounts that are less than are currently being used in insurance settlements, and in some cost-benefit studies for other areas of risk.

TOTAL ESTIMATED COSTS

As shown in the table at the end of the prepared statement, costs for the five foodborne diseases that I examined are estimated at $1.5 to $2.7 billion annually. Three of the five, campylobacteriosis, salmonellosis, and congenital toxoplasmosis—all diseases that have been mentioned by the previous two speakers—each has estimated annual costs of at least $200 million apiece. Trichinosis and beef tapeworm each have estimated costs of less than $4 million. I will now discuss how these cost estimates were obtained for the three most important diseases.

SALMONELLOSIS

At the time of my 1986 study, the Centers for Disease Control [CDC] received approximately 40,000 reports of salmonellosis cases each year, and estimated that of the 2 million U.S. cases, 2,000 caused death.

Based on the reported age distribution of individuals who died from salmonellosis, the estimates of lost productivity from death range from $85,800 to $351,500 per individual, using those two estimation methods I have previously mentioned.

In 1978, Cohen and other CDC researchers published costs for a Colorado outbreak of salmonellosis and reported average costs of $1,290—updated to 1985 dollars. The total included $938 for medical costs, wage or productivity losses of $289 per case, and miscellaneous costs of $62. The CDC Colorado study is the most extensive available. For the cost calculations cited here, the Colorado estimates were assumed to represent the average costs incurred by the 40,000 salmonellosis cases that are reported annually.

Assigning costs to unreported cases is most subjective. To be on the low side, the rest of the estimated 2 million annual cases of salmonellosis are assigned to the mild severity category. The Colorado CDC study estimated an average of $230 per case—updated to 1985 dollars—associated with the unreported cases.

Summing up, the total medical costs and lost productivity, including loss of life, for foodborne salmonellosis in the United States is estimated to be $553 to $988 million annually. This is based on

assuming that 82 percent of the cases of salmonellosis are from a foodborne origin.

Productivity losses represent about 95 percent of the total costs because of the large number of mild cases which do not incur professional medical costs and because of the productivity losses resulting from death.

CAMPYLOBACTERIOSIS

The second disease is campylobacteriosis, and in the interest of time I am going to have to skip that, but the method of calculating it is very similar to salmonellosis. But in summing up for campylobacteriosis, the annual medical costs and productivity losses associated with campylobacteriosis are estimated from $723 million to $1.4 billion. As with salmonellosis, productivity costs are the most important.

CONGENITAL TOXOPLASMOSIS

The third disease is congenital toxoplasmosis. Pregnant women who become infected with toxoplasmosis risk a 20 to 40 percent chance the fetus will be infected. Surviving babies are likely to suffer eye damage and the risk of mental retardation is increased. A study by Wilson and Remington estimated that 3,300 babies are born each year with the infection. Other researchers, such as Walls and Frenkel, suggest that 50 to 75 percent of the infections are associated with foodborne causes. The Wilson and Remington study assigned a total annual cost of $430 million to the infection in babies. The proportion resulted from foodborne causes is $215 to $323 million annually. These are conservative estimates since they do not include the child's potentially reduced lifetime earnings and productivity. Costs of disease in adults are not estimated.

SUMMARY

In summary, the annual U.S. medical costs and productivity losses for the five foodborne diseases, which I have examined, are estimated from $1.5 to $2.7 billion. Trichinosis and beef tapeworm infections incur relatively minor costs, but salmonellosis, campylobacteriosis and congenital toxoplasmosis each cost hundreds of millions of dollars.

These estimates should be only interpreted as indicative and not as precise dollar amounts. Documented costs are made from small samples, hence the extrapolations to nationwide totals are subject to large errors. Also, the vast majority of cases estimated by CDC fall into the mildly severe category, where actual reporting is very limited.

For several reasons, these estimates can be presumed to underestimate the total costs associated with these five foodborne illnesses. For example, the costs associated with chronic complications were not included, and that is an area in which Dr. Archer has a great deal of expertise.

At this point I will cut off my remarks. Thank you, Mr. Chairman. I will be glad to answer your questions.

[The prepared statement of Dr. Roberts follows:]

STATEMENT BY TANYA ROBERTS, AGRICULTURAL ECONOMICS
ECONOMIC RESEARCH SERVICE, DEPARTMENT OF AGRICULTURE
BEFORE THE SENATE COMMITTEE ON AGRICULTURE NUTRITION
AND FORESTRY, JUNE 4, 1987

Mr. Chairman and Members of the Committee, I appreciate the opportunity to provide you with some estimates of the costs of certain foodborne diseases.

It is important to emphasize that it is difficult to estimate costs associated with particular diseases, and usually requires the use of subjective assumptions. Also, researchers differ on the appropriate conceptual cost model to use. Some argue that the correct method is to add up all the direct and indirect costs to individuals, businesses and public agencies resulting from incidence of disease. Others suggest that a more appropriate method to determine the true cost is to measure how much individuals and society are willing to pay to prevent various levels of disease incidence. In addition to differences in methods, the incidence of foodborne disease is difficult to measure because mild cases tend not to be reported or accurately identified.

For these and other reasons, the economic costs due to foodborne diseases are just beginning to be evaluated. Today, I will summarize some estimates of costs for five foodborne diseases: salmonellosis and campylobacteriosis which cause intestinal disturbances; congenital toxoplasmosis which can cause eye problems and mental retardation in fetuses; trichinosis which can cause a variety of ailments; and beef tapeworm which causes discomfort. The estimated direct and indirect costs associated with incidence of disease are used to measure costs.

In my comments, I first will discuss the general procedures used to calculate costs and provide specific estimates for the three most important diseases: salmonellosis, campylobacteriosis and congenital toxoplasmosis. Cost estimates associated with trichinosis and beef tapeworm are presented but not discussed in detail. If the Committee is interested, a more complete description of the procedures and estimates are published in the Proceedings of the 90th Annual Meeting of the U.S. Animal Health Association.

Procedures

Economic costs associated with the foodborne diseases can be characterized as: direct medical costs for physicians, medication, laboratory tests, and hospitalization; costs of lost productivity

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associated with time away from work; value of an individual's pain and suffering; costs of being denied the ability to engage in nonwork related activity; costs associated with attempts to avoid the disease; costs incurred by industry to either prevent disease and/or react to an actual outbreak; and costs of public health surveillance. Cost estimates presented here today are partial because they include only two of the seven cost components, direct medical expenditures and lost productivity. Methods are being developed for evaluating other cost components.

Two types of information are critical to the estimation of costs associated with foodborne illnesses: first, estimates of the number and severity of cases which occur in the population in a typical year; and second, estimates of the average medical and lost productivity costs associated with individual cases of various severity. Since symptoms of foodborne disease range from minor discomfort to death, it is difficult to determine the total number of cases. Epidemiological case studies have shown that incidences of foodborne diseases are greatly underreported because many cases are either not correctly diagnosed or are mild enough to not require professional medical care.

For my 1986 study, "The Economic Losses Due to Selected Foodborne Diseases," I defined three disease severity levels: mild, moderate and deadly. Mild cases represent a day or two of illness and are largely unreported. The number of mild cases, therefore, is inferred from reported cases and expert opinion. Moderate cases are defined as those incidences where the patient is ill for 1 - 2 weeks and seeks professional medical care. Moderate cases often are reported. In some circumstances, foodborne disease can result in death. Although frequently reported, some deaths that result from foodborne disease are incorrectly attributed to other causes. Thus, cost estimates must include reported deaths as well as an estimate of incorrectly diagnosed or unreported deaths.

In the cases where death occurs, the cost estimation process includes appropriate methods for valuing life. I have relied on two academic measures of value of life. The first is Dolan, Hodgson, and Wun's estimate of the individual's contribution to future production, discounted to the year of death. This procedure results in a comparatively low dollar amount. The second measure from Landefeld and Seskin includes values for nonlabor income, risk aversion, and a higher

valuation on future earnings than the first measure. This measure gives the high end of the cost of death range. It should be noted that even the high measure provides dollar amounts somewhat less than currently being used in insurance settlements and cost/benefit studies for other areas of risk.

Total Estimated Costs

As shown in the table at the end of this statement, costs for the five foodborne diseases are estimated at $1.5 - $2.7 billion annually. To place these figures in perspective, it is helpful to look at some other annual costs. Rice, Hodgson and Kopstein estimate the annual cost of neoplasms in the United States at $50.5 billion (1980 dollars). Drunk driving accidents are estimated to cost $13.2 billion (1982 dollars).

To return to the five foodborne diseases cited previously, three of the five--campylobacteriosis, salmonellosis, and congenital toxoplasmosis --each has estimated annual costs of at least $200 million. Trichinosis and beef tapeworm each have estimated costs of less than $4 million.

I now will discuss how cost estimates were obtained for the three most important diseases.

Salmonellosis

At the time of my 1986 study, the Centers for Disease Control (CDC) received approximately 40,000 reports of salmonellosis cases each year, and estimated that of the 2 million U.S. cases, 2,000 caused death. To return to my earlier comparisons, roughly 350,000 deaths from neoplasms are reported annually, and drunk driving fatalities claim an estimated 23,500 victims each year.

(It must be noted that since the 1986 study, the National Academy of Sciences (NAS) issued its "Poultry Inspection" report which cites higher estimates for the total number of cases of salmonellosis and campylobacteriosis and lower estimates for deaths than were provided by CDC for my study. Based on a U.S. population of 240 million, the NAS salmonellosis estimates are 3.6 - 4.8 million cases annually. Of these, 3,360 cases were estimated to result in hospitalization and the estimated number of deaths ranged from 48 - 480. For campylobacteriosis, NAS estimates 2.4 - 4.8 million cases annually. Of these, 16,800 hospitalizations are estimated to result. NAS judged that the data were inadequate to estimate the number of deaths.)

Based on the reported age distribution of individuals who died from salmonellosis, the

estimates of lost productivity from death range from $85,800 - $351,500 per individual for the two estimation methods.

In 1978, Cohen and other CDC researchers published costs for a Colorado outbreak of salmonellosis and reported average costs of $1,290 (1985 dollars). The total included $938 for medical costs, wage or productivity losses of $289, and miscellaneous costs of $62. The CDC Colorado study is the most extensive available. For the cost calculations cited here, the Colorado estimates were assumed to represent the average costs incurred by the 40,000 cases reported annually.

The rest of the estimated 2 million annual cases are in the mild severity category. Assigning costs to unreported cases is most subjective. The Colorado CDC study estimated an average cost of $230 per case (using 1985 dollars) associated with unreported cases.

Total medical costs and lost productivity, including loss of life, for foodborne salmonellosis in the United States is estimated to be $553 - $988 million annually. Cohen and Gangarosa estimate that foodborne sources account for 82 percent of the known causes of salmonellosis.

Productivity losses represent about 95 percent of the total costs because of the large number of mild cases which do not incur professional medical costs and because of the productivity losses resulting from death.

Campylobacteriosis

Surveillance data for Campylobacter jejuni food poisoning is scarce; however, recent studies suggest that it is more common than Salmonella as a cause of intestinal flu-like disease. CDC estimates there are 2.1 million cases annually in the United States. CDC also estimates that 2,100 people die each year as a result of the disease. Extrapolations from a 1985 Denver study by Smith and Blaser suggest estimates of 57,340 cases per year of moderate severity in the United States. Extrapolations from a 1984 Seattle study suggest a level of 168,025 moderately severe cases per year. These two estimates provide upper and lower bounds on moderate severity. The remaining cases are assumed to be of mild severity.

The Seattle study found that duration of illness and likelihood of hospitalization are somewhat higher for campylobacteriosis than for salmonellosis, but actual medical costs and productivity loss data are not available. The cost estimates presented here assume that for given

severity levels the medical costs and costs of productivity loss average the same as for salmonellosis cases of similar severity. Thus, these estimates should be considered conservative and tentative. Based on this assumption, annual medical costs and productivity losses associated with campylobacteriosis are estimated from $723 million to $1.4 billion. As with salmonellosis, productivity costs are most important.

Congenital Toxoplasmosis

Pregnant women who become infected with toxoplasmosis risk a 20 - 40 percent chance the fetus will be infected. Surviving babies are likely to suffer eye damage and the risk of mental retardation is increased. A study by Wilson and Remington estimated that 3,300 babies are born each year with the infection. Other researchers, such as Walls and Frenkel, suggest that 50 - 75 percent of the infections are associated with foodborne causes. The Wilson and Remington study assigned a total annual cost of $430 million to the infection in babies. The proportion resulting from foodborne causes is $215 - $323 million annually. These are conservative estimates since they do not include the child's potentially reduced lifetime earnings and productivity. Costs of disease in adults are not estimated.

Summary

Annual U.S. medical costs and productivity losses for five foodborne diseases are estimated from $1.5 - $2.7 billion. Trichinosis and beef tapeworm infections incur relatively minor costs but salmonellosis, campylobacteriosis and congenital toxoplasmosis each cost hundreds of millions of dollars.

These estimates should be only interpreted as indicative and not as precise dollar amounts. Documented costs are from small samples, hence the extrapolations to nationwide totals are subject to large errors. Also, the vast majority of cases estimated by the CDC fall in the mildly severe category where actual reporting is severely limited.

For several reasons, these estimates can be presumed to underestimate total costs associated with these five foodborne illnesses. Estimated costs of congenital toxoplasmosis do not include the economic costs of reduced productivity. Costs of adult infections of toxoplasmosis were not estimated due to lack of sufficient data. For all five diseases, costs associated with chronic complications were not included, and indirect costs of lost nonwork activity and preventive costs were not measured.

Human illness costs represent only part of the economic costs associated with foodborne diseases. German researchers, such as Krug, have estimated that the costs of reduced output from salmonellosis in cattle, calves, and poultry is greater than the human illness costs. Todd found that food processor and food service industry costs associated with an outbreak and the resulting reduction in demand, product recall, plant closings, and product liability suits may be as significant as the human illness costs.

Thank you, Mr. Chairman. I will be glad to answer your questions.

Table 1. Medical Costs and Productivity Lost due to Selected Foodborne Diseases, United States, 1985 dollars

Foodborne Disease	Cost Estimates Low	High
	Million Dollars	
Salmonellosis	553.0	988.0
Campylobacteriosis	723.0	1,399.0
Trichinosis	2.2	3.5
Congential toxoplasmosis	215.0	323.0
Beef tapeworm	0.1	0.1
TOTAL	$1.5 - $2.7 billion	

Assuming that 100 percent of the cases of trichinosis, beef tapeworm and campylobacteriosis have foodborne sources. Salmonellosis is based on 82 percent of cases from foodborne sources while congential toxoplasmosis is based on half (low estimate) to three-fourths (high estimate) of cases from foodborne sources.

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(The article, "The Economic Losses Due to Selected Foodborne Diseases," by Tanya Roberts, Ph.D, taken from the Proceedings of the 90th Annual Meeting of the United States Animal Health Association, Louisville, Kentucky, 1986, follows:)

THE ECONOMIC LOSSES DUE TO SELECTED FOODBORNE DISEASES

Tanya Roberts, Ph.D.*

The prevalence of some foodborne infectious diseases is thought to be increasing in developed countries. The reported incidence of salmonellosis has doubled in the United States in the last sixteen years to about 40,000 cases annually.[1] The Illinois milk outbreak caused salmonellosis in more than 16,000 people in the spring of 1985. Public health officials estimate that 2 million cases of salmonellosis may occur annually in the United States (Carter Center). Campylobacteriosis, a similar intestinal disease, has an estimated incidence of 2.1 million cases annually.

The causes of this increasing incidence of foodborne disease are diverse. Some of the contributing factors include the following. There is a greater concentration of animals in larger production units, which permits easier transmission of disease from one animal to the other. There is considerable geographic movement of animals and birds that can spread disease across the countryside. Today the use of improperly processed animal by-products and wastes in animal feeds can introduce and perpetuate disease cycles. Concentration of animal slaughter in fewer and larger plants increases the possibilities for cross-contamination between carcasses (Schwabe, pp. 552–53; Snoeyenbos). An increased number of distribution stages and eating away from home means more mass production of food and the greater inherent possibilities of improper heating and refrigeration—two of the most common contributors to foodborne disease in meat and poultry (Bryan). People are traveling more and eating more exotic foods and being exposed to a greater variety of foodborne hazards. Finally, the organisms themselves have been evolving. They are adapting to modern food processing and are more able to survive (Archer). Also, they are developing resistance to human drug therapies (Holmberg, et al.).

Methodology

Foodborne disease costs are just beginning to be evaluated. A consistent methodology is still being developed for evaluation of many components, such as the value of life and the value of discretionary time lost while ill. Here I examine partial costs for five foodborne diseases: salmonellosis, trichinosis, toxoplasmosis, campylobacteriosis and beef tapeworm. Individual's illness costs are estimated for medical expenses and lost productivity. A more complete accounting would also include the individual's

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[1]U.S. civilian population growth was less than 20% from 1986 to 1984.

Taken from the Proceedings of the 90th Annual Meeting of the United States Animal Health Association, Louisville, Kentucky, 1986.

pain and suffering, discretionary time lost, averting behavior,[2] industry costs, and public costs (table 1).

Medial Costs of Illness

(a) All diseases are typically underreported.[3] Epidemiologists have found that only 1 in 75 salmonellosis cases are reported (Smith and Blaser, 1985). Rather than just rely on reported cases, estimates of the total U.S. incidence are used whenever possible.

(b) The severity of illnesses caused by the diseases considered here vary from essentially unnoticeable to life-threatening. The incidence and severity of illness depends upon the number of organisms or amount of toxin encountered and in what foods, the pathogenicity of the organism or toxin, and the strength of the individual's immune system in defending against the pathogenic agents (figure 1). However, fatalities can occur in relatively normal human adult hosts (Smith and Blaser, 1985). Where possible, costs have been estimated for three disease severity levels—mild, moderate, and deadly. Mild cases represent a day or two of illness and moderate cases a week or two of illness.

(c) Secondary data sources are used to derive 1985 cost estimates. Often these costs are estimated from surveys of people involved in an outbreak of foodborne disease.

Productivity Lost

Days lost from work are the typical measure of lost productivity and this time is valued at the individual's wage, or the average wage rate if the individual's wage is unknown.[4] The United States Department of Labor's Bureau of Labor Statistics reports "average weekly earning of production or nonsupervisory workers on private nonagricultural payrolls."

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[2]Averting behavior costs (behavior designed to avoid or reduce the risk of illness) can also be a significant cost item. A recent Resources for the Future study of the contamination of a water supply found the public will pursue a variety of measures to avoid illness—boiling water, travelling to another community to obtain water, and purchasing bottled water (Harrington, et. al., 1985).

Averting behavior by the public can result in diet and consumption expenditure changes that affect sales and revenues of the involved industry. An opinion survey by the National Pork Producers Council found that 40 percent of the surveyed consumers claimed that they had reduced their consumption of pork because of health concerns about salt and 17 percent claimed decreased poultry consumption because of disease concerns (Weise Research Associates, 1984). Of course, opinion surveys by themselves do not provide empirical evidence of actual reductions in consumption and impacts upon industry revenue.

[3]Foodborne diseases are even more likely to be underreported because the reporting by physicians is voluntary, often the cases are mild and of limited duration so physicians may not be seen, the suspect food is seldom available to be tested for pathogens, and many foodborne pathogens have only recently been recognized and are not routinely tested (Archer, 1984, p. 321).

[4]Some economists have also looked at reduced productivity while sick, but still reporting for work. Others have also attempted to make adjustments for heightened productivity when reporting back to work and attempting to catch up with the work backlog. Here these fine tunings are ignored and the average wage rate is used.

Evaluation of Death

Traditionally, deaths have been evaluated by the human capital method which measures the individual's contribution to productive output. The income stream that would have been produced by the individual is collapsed into a present value for that production at today's prices. The human capital method only places a value on what the individual produces for society. This will be used for the low estimate of the value of life.

From the perspective of the individual and consumer demand theory, a life ought to be valued by what the individual is willing to pay to avoid a particular risk of death. The individual's non-labor sources of income are included as resources to pay for risk reduction along with wages. Even more important are the nonmarket activities that may be of more value to the individual than his/her income lost (table 1). However, there are measurement problems with this method.

A hybrid approach attempts to bridge the gap between the two methodologies (Landefeld and Seskin, 1982). The adjusted willingness to pay/human capital approach includes only measurable economic losses associated with death (i.e., pain and suffering and lost leisure time are excluded). It is based on after-tax income from labor and nonlabor sources, discounts at the individual's rate of return after taxes, and includes risk aversion shown by investment in life insurance, security systems, etc. This will be the high estimate for the value of life.

The human capital and adjusted willingness to pay/human capital methods are age-specific and the values of life for death due to salmonellosis are $85,800 and $351,500 (1985 dollars), respectively (Morrison and Roberts, 1985, p. IV–10). The value of life for death due to helminthiases (trichinae and related worm-like parasites) is $140,000 and $471,000, respectively (Ibid., p. IV–15).

The Cost of Selected Foodborne Diseases

Medical costs and productivity losses caused by five foodborne diseases are estimated here. These diseases are salmonellosis and campylobacteriosis which cause intestinal disturbances, toxoplasmosis which can cause eye problems and mental retardation in fetuses, trichinosis which can cause a variety of ailments, and beef tapeworm which causes discomfort. The omission of other foodborne diseases does not mean they are trivial, only that they are not evaluated here.

Salmonellosis

Typically, *Salmonella*-caused disease is limited to salmonellosis, an acute gastroenteritis which appears 12 to 74 hours after eating contaminated food. Salmonellosis may cause only mild abdominal discomfort with minimal diarrhea lasting less than a day. More acute symptoms of disease include nausea, stomach ache, vomiting, diarrhea, cold chills, fever, and exhaustion. The symptoms of disease normally persist for 2 to 6

days, but in exceptional cases for several weeks. A small percentage (0.1 percent) of the people who contract salmonellosis die from it, and fatality is generally limited to the sick, those taking antibiotics, infants, or the elderly. However, in rare cases *Salmonella*, like some other bacterial and parasitic infections, can cause chronic disease syndromes (see Appendix).

"The enormous reservoir of contaminated food products, especially poultry products, has caused a steady increase in the incidence of salmonellosis" (Berkow, 1982, p. 103). The number of organisms needed to cause disease varies, but as few as 10 cells of *Salmonella* per gram or per milliliter in hamburgers and pasteurized milk have been sufficient (Mulder, 1982, p. 8). The Carter Center for CDC/Emory University estimates 2 million cases of salmonellosis occur in the United States each year (Holmberg, 1985). Foodborne sources account for 82 percent of the known causes (Cohen and Gangarosa, 1978).

The most extensive data on costs come from a CDC investigation of the Colorado outbreak (Cohen, et. al., 1978). The average survey respondent reported costs of $1,290 (in March 1985 $)—medical costs of $938, wage or productivity losses of $289 and miscellaneous costs of $62 (Roberts, 1985). The data from this outbreak are assumed to be representative of all cases reported to CDC. There are roughly 40,000 salmonellosis cases reported annually (Holmberg, 1985).

For those 2,000 salmonellosis patients who are estimated to die, the present value of life is estimated using the two methods discussed and result in values per life of $85,800-low estimate and $351,500-high estimate.

Mild cases of salmonellosis are the remainder of the estimated 2 million cases minus the 40,000 reported cases, or 1,960,000 cases. Costs are not well studied. Some of the unreported cases are as serious as the reported cases, but the bulk are probably much less severe. How much less severe is not known. The mild cases are assumed to have costs of $230 apiece (March 1985 $) which is what Cohen et. al. estimated for unreported case of salmonellosis in the Colorado outbreak.[5]

The medical costs and lost productivity for foodborne salmonellosis in the United States total an estimated $553 to $988 million annually (table 2). Notice that medical costs are much smaller than the productivity losses.

Campylobacteriosis

Surveillance data for *Campylobacter jejuni (C. jejuni)* food poisoning is spotty, but all recent studies indicate it is more common than salmonellosis as a cause of intestinal flu-like disease. The Carter Center of CDC/

[5]The $125 figure reported in the original article is updated using the change in the Consumer Price Index.

Emory University estimates there are 2.1 million cases annually in the United States. A conservative measure of the costs of campylobacteriosis illness would be the salmonellosis costs for illnesses and deaths, given that the duration of the illness and likelihood of hospitalization were somewhat greater for campylobacteriosis in the Seattle study. Campylobacteriosis is generally self-limiting, although like salmonellosis more serious complications can occur, such as colitis, arthritis, and meningitis (Appendix).

Surveys in Denver and Seattle give us two estimates of the number of cases of moderate illness (Smith and Blaser, 1985; Seattle, 1984). Extrapolating to the United States, the Denver study suggests 57,340 moderate cases annually nationwide and the Seattle study suggests 168,025 moderate cases nationwide.

The mild cases are the residual between the 2.1 million annual cases of campylobacteriosis and the low and high estimate for the moderate cases. Like salmonellosis, the case-fatality rate it 1,000 to 1, for an estimated 2,100 fatalities a year.

The medical costs and productivity losses associated with campylobacteriosis are estimated at between $723 million to $1.4 billion annually (table 3). Again, the productivity costs are more important than medical costs.

Trichinosis

The decline in human cases of trichinosis has been dramatic in the U.S., while in Germany the disease has all but disappeared, largely because they inspect for it (Pawlowski, 1981). Trichinosis varies from asymptomatic illness to death depending largely on the numbers of live larvae ingested which have not been killed by cooking. After offspring are produced in the small intestine, they migrate throughout the body, lodge in the muscles, and may produce a wide variety of ailments. Consequently many cases of trichinosis are undiagnosed because they are so mild or misdiagnosed because of the diversity of symptoms (Zimmerman, Steele and Kagan, 1973). Autopsy data indicates there may be 100,000 exposures annually (Carter Center, 1985). Most of these cases are successfully fought off by the individual's immune system without causing illness in the individual.

Reported cases of trichinosis averaged 152 human cases annually from 1975–81 (Schantz, 1983). To estimate the unreported cases, there is information from an outbreak occurring at sea. Three cases of trichinosis were reported while 10 other cases were found in the follow-up epidemiological investigation (Singal, Schantz, and Werner, 1976). If this outbreak is representative, then there are 3.3 unreported cases for each reported case of trichinosis (10/3). The estimated number of *unreported* cases of trichinosis is 502 annually (152 reported cases × 3.3). The total becomes an estimated 654 cases annually (152 reported + 502 unreported).

Medical costs and lost wages were estimated for an outbreak of trichinosis affecting seventeen people in 1975 (Potter *et al*, 1976). In 1985 prices the per patient medical cost was $1,860 (Roberts, 1985). Multiplied times the estimated 654 cases resulted in $1.2 million medical cost per year for trichinosis. The wages lost averaged $625 in this outbreak, for a total of $.4 million.

Also, about one death a year is reported for trichinosis. I assume that deaths are underreported just like illnesses and that roughly four deaths a year occur. Evaluating these deaths with the two methods results in a range of $.5 to 1.9 million for the value of lives lost. This gives a total cost range for illness and deaths due to trichinosis of $2.2 to 3.5 million (table 4).

Toxoplasmosis

Another disease arising from consuming undercooked pork, or in rare instances handling of raw pork, is toxoplasmosis. There are only two environmental sources of infection—meat and ingesting soil contaminated with cat feces. Pork and mutton are commonly infected in the U.S. whereas beef or broilers are rarely infected (Ruiz and Frenkel, 1980). Dubey, et. al. (1979) report that several surveys show widespread, nonclinical infections with *Toxoplasma gondii* in U.S. pigs. There are no concrete numbers on the sources of human toxoplasmosis, but public health researchers suggest that pork causes half to three quarters of the U.S. cases.[7] This conclusion was reached by surveying households with cats and taking blood samples to determine whether exposure had occurred. No systematic relationship was found. Instead a relationship was found between adults and exposure, which was attributed to the post-adolescent cultural habit of eating undercooked or raw meat.

If a pregnant woman becomes infected with toxoplasmosis there is a 20–40 percent probability her fetus will be infected. Surviving babies are likely to suffer eye damage and have a probability of mental retardation. An estimated 3,300 babies born every year in the U.S. are infected. Wilson and Remington conservatively estimated the life time cost for 3,300 children born with congenital toxoplasmosis in the U.S. every year was $430 million each year (updated to 1985 prices in table 5). Because babies born every year are exposed to the risk of toxoplasmosis, in a steady state with a constant number of births each year, the annual costs incurred

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[6]While death certificates list the cause of death, the variety of organs affected by trichinosis make the diagnosis difficult unless an autopsy is performed.

[7]Kenneth Walls, Protozoan Disease Branch, CDC suggested 50 percent and Jacob Frenkel, University of Kansas Medical Center, suggested 75 percent (personal communications, 1985). Also, Dubey, Murrell and Fayer report that "ingestion of cysts in undercooked pork infected with *Toxoplasma gondii* is thought to be a major source of *Toxoplasma* infection in human beings" (1984, p. 1941). They also report that *Toxoplasma gondii* can persist in the edible tissue of pigs until slaughter.

would be $430 million each year. Then, the cost of mental retardation and eye problems in babies due to the 50–75 percent of cases caused by pork is estimated at $215 to $323 million annually.[8]

Beef Tapeworm (Taenia saginata)

Eating a larvae that has not been killed by thorough cooking will infect a person. Unless medical treatment is provided, the mature tapeworm will live in the intestine until the death of the individual. Symptoms vary in their intensity and some persons never realize they have a tapeworm. However most people experience symptoms which "... may include nervousness, insomnia, anorexia, loss of weight, abdominal pain, and digestive disturbances" (Benenson, 1975, p. 319). Occasionally, other organs are invaded and serious disorders can occur (Hubbert, McCullock, and Schnurrenberger, 1975, p. 679).

The Carter Center of CDC/Emory University estimates that 1,000 tapeworm cases occur annually in the U.S. Typically the disease symptoms are mild. The cost to remove a tapeworm involves two visits to a doctor, a stool examination, and drug treatment (Roberts, 1983, p. 34). These medical costs and time-off work for the visits to the doctor are estimated at $111 per case (Morrison and Roberts, 1985). The annual cost due to the beef tapeworm, *Taenia saginata*, is conservatively estimated at $.1 million.

Disease Cost Summary

The medical costs and productivity losses for five foodborne diseases associated with raw chicken, pork and beef are estimated to total between 1.5 and 2.7 billion dollars annually in the United States (table 6). Trichinosis and beef tapeworm are relatively minor hazards, but salmonellosis, campylobacteriosis and congenital toxoplasmosis all have impacts in the hundreds of millions of dollars each year. Lost productivity is the greatest cost component, significantly outweighing medical costs. However, these cost estimates omit the chronic complications that can result from all five diseases, ranging from arthritis to blood poisoning to heart disease (Appendix). Also omitted are lost leisure time, travel costs, and averting behavior costs—which could be important for trichinosis, in that people

[8]An estimated 2.3 million cases of toxoplasmosis occur in adults annually. Most cases are mild and fought off by the individual's immune system. However, toxoplasmosis can cause three types of disease syndromes in adults. A common infection of the lymph system; the occasional, more severe infection of the lymph system plus another organ such as the brain, heart, lung, liver or skeletal muscles; and finally a rare life threatening, generalized toxoplasmosis which is usually associated with immunological deficiency.

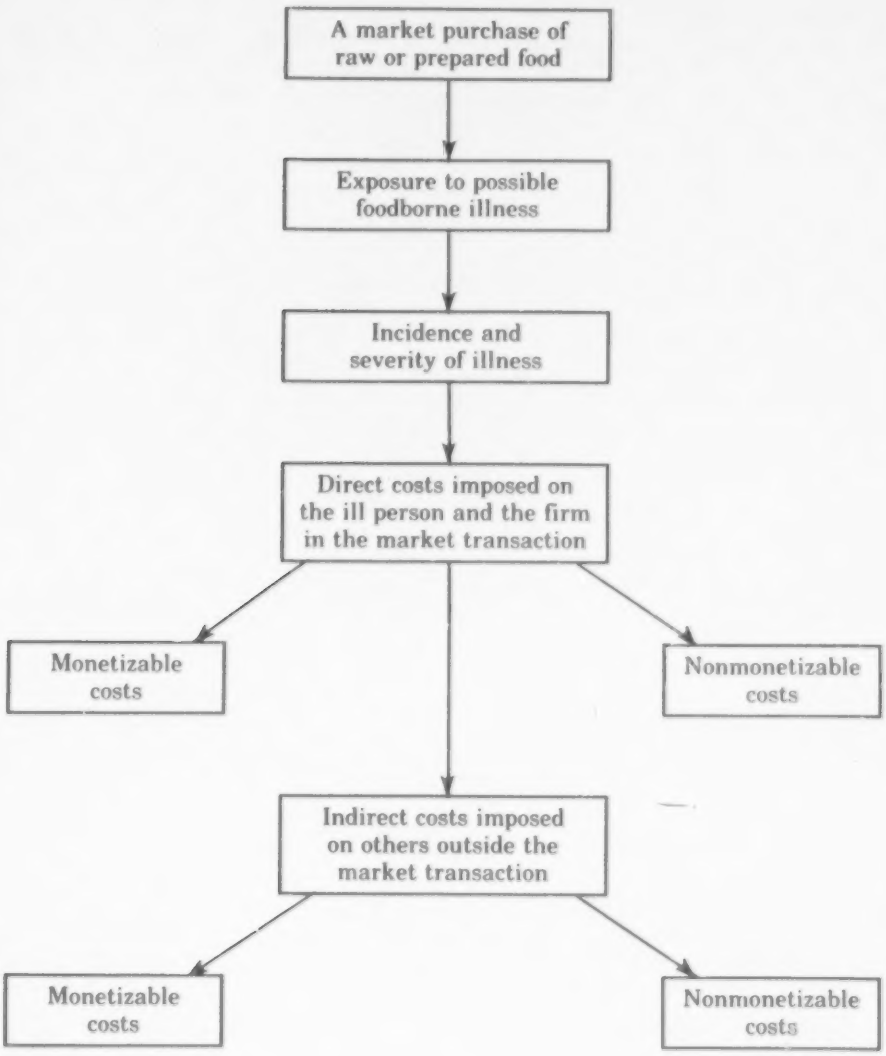
Once exposed to toxoplasmosis, the individual is likely to be left with cysts embedded in tissue for the rest of his/her life. If the individual's immune system fails because of old age, immunosuppressive diseases such as AIDS or cancer, immunosuppressive drugs, or if the cysts are mechanically broken, an acute phase of the infection begins again (Fayer and Dubey, 1985, p. 57).

The benefit of preventing these adult cases is likely to be substantial, but not quantified at this time.

may either overcook the meat or avoid it altogether. Pain and suffering are underestimated or omitted for the ill person and other family members and friends.

These estimates also exclude the impact on industry at both the farm level and the retail level. German researchers have estimated that the costs of reduced output try is greater than the human illness costs (Krug). Todd found the food processor's or restaurant's costs associated with an outbreak and the ensuing reduction in demand, product recall, plant closings, and product liability suits may be as significant as the human illness costs. For salmonellosis, the estimates presented here exclude the possibilities of cross contamination from chicken and beef to other cooked or raw foods (Bryan, Ayers, and Kraft).

Figure 1. Direct and Indirect Foodborne Illness Costs


A market purchase of raw or prepared food
Exposure to possible foodborne illness
Incidence and severity of illness
Direct costs imposed on the ill person and the firm in the market transaction
Monetizable costs
Nonmonetizable costs
Indirect costs imposed on others outside the market transaction
Monetizable costs
Nonmonetizable costs

77-794 0 - 88 - 4

Table 1. Social Costs of Foodborne Illness

Costs to Individuals	Industry Costs
Medical Costs	Product recall
Income or productivity loss	Plant closing and cleanup
Pain and suffering	Product liability costs*
Leisure time lost	Reduced product demand
Averting behavior costs	**Public Health Surveillance Costs**
Risk aversion costs	Costs of maintaining disease surveillance
Child care costs	Costs of investigating outbreak
Travel costs	Costs of cleanup

*In adding up costs, care must be taken to assure that product liability costs to firms are not already counted in the estimated pain and suffering cost to individuals. Recent controversy over the size of liability claims has caused many states and the U.S. Congress to consider limiting liability for noneconomic damages to $100,000 per person.

Table 2. Cost of Foodborne Salmonellosis, U.S., 1985 dollars

Cost Categories	Costs per case	Cases	Total
	dollars	number	million dollars
Medical Costs			
Mild cases	0	1,960,000	0
Moderate cases and death	938	40,000	37.5
Lost Productivity			
Mild cases	230[b]	1,960,000	450.8
Moderate cases	289[c]	38,000	11.0
Deaths:			
Human capital method	85,800		171.6
or	or	2,000	or
Adjusted human capital/ willingness to pay method	351,500		703.0
Miscellaneous costs[a]	62	40,000	2.5
Total			$673 to $1,205 million
Foodborne sources			$553 to $988 million

[a] Miscellaneous costs include transportation, child care, other laboratory tests, finding new jobs.

[b] This productivity loss estimate for mild cases also includes some miscellaneous costs of undetermined amount.

[c] This is a low estimate because it leaves out the value of homemaking.

Table 3. Costs of Foodborne Campylobacteriosis, U.S., 1985 dollars

Cost Categories	Costs per case	Cases	Total Estimate Low	Total Estimate High
	dollars	number	million dollars	
Medical Costs				
Mild cases	0	2,042,660 or 1,931,975	0	0
Moderate cases and deaths	938	57,340 or 168,025	53.8	157.6
Lost Productivity				
Mild cases	230[b]	2,042,660 or 1,931,975	469.8	444.4
Moderate cases	289[c]	55,340 or 166,025	16.0	48.0
Deaths:				
Human capital method or Adjusted human capital/ willingness to pay method	85,800 or 351,500	2,100	180.2	738.2
Miscellaneous[a]	62	57,340 or 168,025	3.6	10.4
Total			$723 to $1,399 million[d]	

[a] Miscellaneous costs include transportation, child care, other laboratory tests, finding new jobs.
[b] This productivity loss estimate for mild cases also includes some miscellaneous costs of undetermined amount.
[c] This is a low estimate because it leaves out the value of homemaking.
[d] Assumes all campylobacteriosis is of foodborne origin.

Table 4. Cost of Foodborne Trichinosis, United States, 1985 dollars

Categories	Cost per case	Cases	Cost
	dollars	number	thousand dollars
Medical Costs	1,864	654	1,216
Productivity losses			
moderate cases	625	654	409
deaths	140,000 or 471,000	4	560 or 1,884
Total			$2.2 to 3.5 million

Table 5. Lifetime Cost for Special Services for 3,300 Children with Congenital Toxoplasmosis Born in the United States Each Year, 1985 dollars*

Service Required	Utilization	Cost of Service
	percentage	million dollars
Yearly ophthalmologic follow-up care	78.0	4
Special schooling for visually handicapped	14.2	68
Special schooling for moderately retarded	7.1	23
Institutional or state-supported foster care for severely retarded	15.1	301
Aid to totally disabled	2.4	33
Total		$430 million*

*This estimate is conservative because it does not include pain and suffering for the babies born with a mental or visual handicap and the suffering of the parents of these babies or the 15 percent who died. In addition, the survivors' medical complications have been underestimated and no adjustment has been made for reduced earning capacity caused by toxoplasmosis. Finally, the income losses due to parents caring for the child instead of working are not included.

Source: Wilson and Remington and updated by author to 1985 dollars using the Consumer Price Index.

Table 6. Medical Costs and Productivity Lost due to Selected Foodborne Diseases, United States, 1985 dollars

Foodborne Disease	Cost Estimates low	high
	million dollars	
Salmonellosis	553	988
Campylobacteriosis	723	1,399
Trichinosis	2.2	3.5
Congenital toxoplasmosis	215	323
Beef tapeworm	0.1	0.1
Total	1,493	2,714

Assuming that 100% of the cases of trichinosis, beef tapeworm, and campylobacteriosis have foodborne sources. Salmonellosis is based on 82% of cases from foodborne sources while congenital toxoplasmosis is based on 1/2 (low estimate) to 3/4 (high estimate) of cases from foodborne sources.

Appendix. Complications Observed After Bacterial and Parasitic Infections Transmitted by Foods

Infection	Complication Caused
Bacterial infections:	
Salmonellosis	cholecystitis, colitis, endocarditis, meningitis, thyroiditis, myocarditis, rheumatoid syndromes, Reiter's disease, splenic abscesses, septicaemia, pancreatitis, osteomyelitis, aortitis
Campylobacteriosis	cholecystitis, colitis, endocarditis, meningitis, arthritis, carditis, septicaemia, erythema nodosum
Parasitic infections:	
Taeniasis	arthritis
Toxoplasmosis	pancarditis, central nervous system disease
Trichinosis	neurological sequelae, cardiac dysfunction

Source: Mossel, 1984, p. 93.

77-794 0 - 88 - 5

Present Value of Life for Salmonellosis Fatalities, 1985

Method	Age	Male			Female			Average Value[3]
		Deaths	Present Value	Total Value	Deaths	Present Value	Total Value	
		number	thousand	dollars	number	thousand	dollars	dollars
Human Capital[1]	0-4	3	88	264	4	77	307	
	5-14	0	159	0	2	139	278	
	15-24	2	300	600	1	241	241	
	25-44	0	371	0	4	238	954	
	45-64	10	189	1,890	6	144	867	
	65 +	20	14	271	27	41	1,107	
		35		3,025	44		3,754	85,800
Adjusted willingness to pay/human capital[2]	0-4	3	1,208	3,624	4	836	3,102	
	5-14	0	1,408	0	2	961	1,922	
	15-24	2	1,655	3,309	1	1,086	1,086	
	25-44	0	1,432	0	4	866	3,462	
	45-64	10	548	5,480	6	410	2,459	
	65 +	20	34	680	27	90	2,443	
		35		13,091	44		14,675	351,500

[1] Data from Dolan, et al, 1980; *Vital*, 1984; Updated to March 1985 dollars.
[2] Data from Landefeld and Seskin, 1982; *Vital*, 1984; Updated to March 1985 dollars
[3] Value calculated by dividing the total values for male and female by total number of deaths.

Present Value of Life for Deaths Caused by Helminthiases

Method	Age	Male			Female			Average Value[3]
		Deaths	Present Value	Sum	Deaths	Present Value	Sum	
		number	thousand	dollars	number	thousand	dollars	dollars
Human Capital[1]	0-4	0	87	0	0	76	0	
	5-14	0	158	0	0	138	0	
	15-24	0	299	0	0	239	0	
	25-44	4	369	1,476	2	237	474	
	45-64	6	188	1,128	0	144	0	
	65+	8	13	104	4	41	164	
				2,708			638	140,000
Adjusted human capital/ willingness to pay[2]	0-4	0	1,202	0	0	821	0	
	5-14	0	1,401	0	0	956	0	
	15-24	0	1,646	0	0	1,080	0	
	25-44	4	1,425	5,700	2	861	1,722	
	45-64	6	545	3,270	0	408	0	
	65+	8	32	256	4	90	360	
				9,226			2,082	471,000

[1] Data from Dolan, et al, 1980; *Vital*, 1984; Updated to February 1985 dollars.
[2] Data from Landefeld and Seskin, 1982; *Vital*, 1984; Updated to February 1985 dollars
[3] Value calculated by dividing the total values for male and female by total number of deaths.

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Senator MELCHER. Thank you. Dr. Menning.

STATEMENT OF EDWARD L. MENNING, D.V.M., M.P.H., EXECUTIVE VICE PRESIDENT, NATIONAL ASSOCIATION OF FEDERAL VETERINARIANS

Dr. MENNING. Thank you, Mr. Chairman.

I will be making a few remarks about some of the evolving knowledge surrounding these agents of foodborne disease that we have been talking about and trying to tie a few of these together.

The major points to remember, I believe, are:

One, that disease from meat, poultry, and seafood is a serious and complex problem.

Two, this is not a new problem though it does have many new facets.

Three, this is not a poultry problem, it is a problem of all raw meats with poultry being but one part.

Four, this is not solely a USDA problem, it is a problem of feed manufacturers, animal producers, slaughtering plants, processing plants, transportation, retail sales, and consumers.

Five, this problem can be reduced and controlled.

We have known most of these diseases in veterinary or human medicine for quite a long time. There are, however, many more agents known today to be producing human disease than we used to think so, and the significant disease in humans is increasing and newer disease entities are emerging, at least from rare, in the past, or not known at all.

Almost all of the human foodborne diseases that have emerged in the past 30 years from meat and poultry are from animals, and often do occur on healthy or subclinically infected animals.

Our knowledge of how these organisms react has also been evolving. Not many years ago we thought that foods kept refrigerated, that is, below 45 °F, would spoil after a period of time, but certainly, disease germs would not multiply in foods at those refrigerated temperatures.

We now know this is not true, especially for Yersinia, Listeria, and about three or four other serious pathogens, which will grow at 39 °F, and above, and quite well.

We also used to believe that it took hundreds of thousands, if not millions of these organisms, to cause disease in a healthy adult. We now know that as low as 100 organisms of certain species of salmonella at least, and as low as 100 Listeria organisms can produce disease in healthy adults. Many persons had thought that most foodborne disease was only a little diarrhea among friends, or as another person said, "a cathartic that is occasionally good for you." We now know that the diarrhea by itself is not little, either from a disease entity or an economic factor. We also know, as Dr. Archer has much information on, that the chronic or long-term effects may be much more severe than the acute disease itself.

There has even been changes in the population at greatest risk, that is, those who would most easily contract these diseases and then be most severely affected. The extremely young are still in this group, but more children are surviving with other debilitating diseases, with our modern medicine, than used to, and, hence, they

are more susceptible to these diseases. The elderly are still in this group; however, they are surviving in far greater numbers, and they also are surviving with other debilitating conditions that make them even more susceptible to these diseases than used to be. The already debilitated who are not young or elderly, also in great numbers, and also are rapidly increasing due to immunocompromising factors, such as antirejection drugs used in transplant surgeries, chemotherapies for cancer patients and of course AIDS.

There has been a revolution in the potential for disease from farm or sea to your table. On the Ohio farm where I was raised, we would go out to the henhouse, catch a chicken—which had eaten only food from our farm—kill it, plunge it into boiling water, remove the feathers, carefully remove the viscera, wash it, and immediately "cook the hell" out of it. If the bird were diseased, and anything escaped cooking, only my family would have become ill. Since then there has been a massive technological change in every stage of handling fresh meat from farm to table, except in control of pathogens for which there has been a concomitant standstill or even backsliding, in my opinion.

Animals are now fed contaminated feeds from all around the country. They are raised in monstrous close-quartered numbers, are commingled in feedlots from around the country, are further commingled at slaughter plant holding pens, then are further crosscontaminated in the slaughter plants, be they beef, pork, or poultry. Production lines go so fast that the equipment furthers the accidental fecal contamination. Water temperature in the scald tanks is so low that it essentially kills nothing. All this is then further crosscontaminated by the cutting up, grinding up, and mixing that is now common. Finally, these mixed-up products are refrigerated, shipped, and received by the retail outlets sometimes under variable temperature conditions. They then are held and sold fresh for many days. These products are now being preserved more with freezing, that keeps the bacteria alive, lower heating temperatures, using less salt, less acid, less sugar, less nitrites, cooked spottily in microwaves, being cooked rare or not being cooked at all.

Making the above more of a threat is the actual decreased education of the American consumer as to what dangers lurk in the shadows of their meat cases. Most people, from my own observations and questions, have no idea that meats, poultry, and seafood should be considered already contaminated with one or more serious pathogens, which will rub off on their hands, sinks, counters, cutting boards, knives, et cetera, and then contaminate the next item touched. USDA pamphlets very beautifully define how you should keep from contaminating your meat at home; however, they do not even intimate that the raw products are probably contaminated when bought, and thence how you should protect yourself from them at home.

What has allowed all of this to happen? That is, little action or inaction on foodborne illness control. This has evolved over the years and I have addressed several specifics in the prepared statement, but, by and large it has been due to apathy, lack of interest by a lot of people, as well as reduced funding, though I would say the reduced funding, in my opinion, has been less of a cause than

the apathy in all groups from medics on through to poultry processors.

Responsibilities have also, for all of these charges, become very intertwined, so that, really, very few people know who is in charge of control

The USDA has played a part in this apathy also. They are by no means the major culprit, and, indeed have done many things right, just not all the right things. They have very effectively removed diseased animals from the food chain, and they have very effectively protected the safety of cured, cooked, or ready-to-eat products. They have performed excellent studies on all phases of speeding up production, many areas of fraud protection, and they have, with great scientific knowledge, developed some species tests that now tell us whether there is chicken meat in our beef hotdog. They, however, have always considered most of these diseases as normal contaminants. If the organism is unseen and/or contaminated from the animal species it is a normal contaminant, and hence, not subject to control. This philosophy must be changed.

In conclusion, each year we have a public health problem causing millions of cases of disease, hundreds of thousands of hospitalizations, thousands of deaths, thousands of deformed children, hundreds of thousands of chronic joint, nutritional, enteric or other diseases. This is a problem from all meats, poultry, and seafoods—poultry is probably not even the major problem. This is not a salmonella problem and not just a fecal problem. There are many diseases that have been alluded to today that are equally important to salmonella. This relates to many agencies and industries, and, again, USDA is not the only major player in this game.

The present cost for inspection of meats is only $1.60 per American per year in the United States. That includes the entire FSIS budget, and that is for all meat, poultry, inspected, slaughtered in the United States, exported from the United States, and imported into the United States. I think that is an extremely good cost-benefit ratio.

We have heard a lot today about diseases. Some will say that there is little that can be done. Others believe much can be done now and for little cost. We must start now, now is already too late. I believe we have the know-how to make a major dent in reducing these diseases immediately. If we do not feel we have the knowledge, we could ask Sweden to help, since they have essentially eradicated salmonella from their food supply.

Thank you, Dr. Melcher.

[The prepared statement of Dr. Menning follows:]

STATEMENT

OF

EDWARD L. MENNING, D.V.M., M.P.H.

NATIONAL ASSOCIATION OF FEDERAL VETERINARIANS

BEFORE THE

COMMITTEE ON AGRICULTURE, NUTRITION AND FORESTRY

UNITED STATES SENATE

JUNE 4, 1987

Witness appearing before the committee:

Edward L. Menning, D.V.M., M.P.H.

Executive Vice President

of the

National Association of Federal Veterinarians

Dr. Menning received his Doctor of Veterinary Medicine from The Ohio State University in 1955 and a Masters in Public Health from the University of Michigan in 1964. He is board certified by the American College of Veterinary Preventive Medicine. He retired from the U.S. Air Force in 1980 as the Assistant Surgeon General for Veterinary Services.

He presently serves as the chief executive officer of NAFV, an association representing about 3000 federally employed veterinarians.

EVOLVING KNOWLEDGE

OF

MEAT/POULTRY FOOD-BORNE PATHOGENS

Mr. Chairman and members of the committee, the National Association of Federal Veterinarians appreciates the opportunity to present information that may help to emphasize the need to control better the agents of human disease that are spread in great part by meat, poultry and seafood.

The major points to remember are: 1) disease from meat/poultry/seafood is a serious and complex problem; 2) this is not a new problem though it does have new facets; 3) this is not a poultry problem, it is a problem of all raw meats with poultry being but one part; 4) this is not solely a USDA problem, it is a problem of feed manufacturers, animal producers, slaughtering plants, processing plants, transportation, retail sales and consumers; and 5) this problem can be reduced and controlled.

We have known most of these diseases in veterinary or human medicine for a long time. Salmonella spp. were first isolated from a food-borne outbreak in 1888, Campylobacter spp. has been known as a serious disease in animals and rare (?) in humans since the late 1890's, Listeria spp. was recognized as a serious disease in sheep and rare (?) in humans since 1925, Toxoplasma spp. was first isolated in 1909 in animals and 1923 in humans--I could go on. We have of course learned many new features of

these diseases since their discovery. Campylobacter was not considered a significant human pathogen until the mid 1970's and is now the leading cause of human bacterial gastroenteritis in the U.S. Listeria were not considered significant to humans until the 1980's. I was taught that the only significance of Escherichia coli was as nonpathogenic indicators of fecal contamination, but we now know of at least four strains that produce serious human disease, one of which causes a hemolytic uremic syndrome that is often fatal. In 1969, the life cycle of Toxoplasma was discovered to involve cats as the primary host which led to its significance in food animals and humans, especially to pregnant women. Aeromonas hydrophila, now is being recognized as a significant food-borne disease entity. Recently Aeromonas was observed by the Agriculture Research Service to be in a variety of animal-based foods and were found to be probably pathogenic. This suggested that these foods are likely to be a significant source of A. hydrophila gastroenteritis. There are many more agents that are known to cause significant disease in humans and more are emerging. Almost all of the human food-borne diseases that have emerged in the past 30 years or more are from animals and occur often on healthy or subclinically diseased animals.

Our knowledge of how these organisms react has also been evolving. Not many years ago we thought that foods kept refrigerated (below 45 ^{o}F) would spoil after a period of time but

that there was no significant danger of disease germ growth while at that temperature. Yersinia spp., a significant cause of disease, including chronic rheumatoid problems and others mentioned by Dr. Douglas Archer, grows well at 40 ° F and above. Listeria spp., the cause of severe disease especially of pregnant women and their fetuses grows well at 39 ° F and above. Also Clostridium botulinum, type E grows above 38 ° F, Vibrio parahaemolyticus above 43 ° F, enterotoxigenic E. coli above 39 ° F and Aeromonas hydrophila above 40 ° F. Cheese aged over 60 days recently was considered safe from living pathogens, we now know Salmonella and others do survive far longer than 60 days. We also used to believe that it took hundreds of thousands if not millions of these organisms to cause disease in a healthy adult. We now know that as low as 100 organisms of some Salmonella spp. can cause disease. As low as 100 Listeria will cause disease in about 10% of persons exposed and other infective doses are slowly being revised downward. Many persons had thought that most food-borne diseases only caused "a little diarrhea among friends" or were even "a cathartic that is occasionally good for you." We now know that the diarrhea by itself is not little either from a disease entity or an economic factor. We also know, as has been testified to, that the chronic or long-term effects may be much more severe than the acute disease itself. We now realize that many of these organisms become resistant to antimicrobials from animal feeds, treatment of animals or treatment of humans. Though these antimicrobially-resistant bacteria do not seem to be

more invasive nor have different growth patterns, their resistance does pose new complications for treatment of enteric pathogens. Their growth and production of new disease in people who are on antibiotics for some other reason and their lack of response if they are treated with a drug to which they're resistant prolongs recovery at best.

There has even been changes in the population at greatest risk, i.e. those who would most easily contract these diseases and then be most severely affected. The extremely young are still in this group but more children are surviving with other debilitating diseases and hence even more susceptible. The elderly are still in this group but their overall numbers are rapidly increasing and they too are surviving with many more other weakening conditions. The already debilitated who are not young or elderly also still exist and their numbers also are rapidly increasing due to immunocompromising factors such as antirejection drugs used in transplant surgeries, chemotherapies for cancer patients and of course AIDS. All of the above categories are much more likely to get all of the diseases we're discussing, as well as to be more severely infected with a high mortality. This is not just supposition but has been shown in studies of various groups of AIDS patients in which high percentages of their secondary infections have been from numerous zoonoses, animal diseases transmitted to humans, some of which are as yet uncommon enough that they haven't been mentioned

today, such as avian tuberculosis, cryptosporidiosis, etc. All of these factors added to an increasing population dictate that unless something is done, more and more people are going to become ill, have severe chronic affects, or die.

There has been a revolution in the potential for disease from farm or sea to your table. On the Ohio farm where I was raised we would go to the henhouse catch a chicken (which had eaten only from from our farm), kill it, plunge it into boiling water, remove the feathers, carefully remove the viscera, wash it, and immediately "cook the hell" out of it. If the bird were diseased and anything escaped cooking, only my family would have become ill. Since then, there has been a massive technological change in every stage of handling fresh meat from farm to table, except in control of pathogens for which there has been a concomitant standstill or even backsliding. Animals are now fed contaminated feeds from all over the country, are raised in monstrous close-quartered numbers, are commingled in feed lots from around the country, are further commingled at slaughter plant holding pens, then are further crosscontaminated in the slaughter plants (beef, pork, chicken, etc.). Production lines go so fast that the equipment furthers the accidental fecal contamination. Water temperature in the scald tanks is so low that it essentially kills nothing. All this is then further crosscontaminated by the cutting up, grinding up and mixing that is now common. Finally these mixed-up products are refrigerated,

shipped, and received by the retail outlets sometimes under variable temperature conditions. They then are held and sold fresh for many days. These products are now being preserved more with freezing that keeps bacteria alive, lower heating temperatures, using less salt (ARS scientists have just shown that Listeria is easily killed by salt after exposure to sublethal heat or acidification thus there is a potential for increased survival as salt levels decrease), less acid, less sugar, less nitrites, cooked spottily in microwaves, being cooked rare or not being cooked at all. This problem begins, at least in a small way, on the farm. It proceeds to be compounded and exacerbated with each step leading to the ultimate consumer who indeed can make it worse. Now there are proposals to retail more refrigerated meats that have been only partially cooked along with the rare duckbreast salad, sushi, and steak tartare. A short shelf life is a consumer's protection and as shelf lives are extended so are dangers. Add to this the unknowns of the newly packaged ethnic and foreign foods being imported. Finally add the potential for genetically engineered foods to further complicate the future picture with lowered acid in tomatoes, etc., etc. Making the above more of a threat is the actual decreased education of the American consumer as to what dangers lurk in the shadows of their meat cases. To this day most people cook pork well because they have been educated about trichinosis. Most people, from my own observations and questions, have no idea that meats, poultry and seafood should be considered already

contaminated with one or more serious pathogens which will rub off on their hands, sinks, counters, cutting boards, knives, etc. and then contaminate the next item touched. USDA pamphlets which portray quite well that meat makes a good growth media for disease germs and which very well defines how you should keep from contaminating it at home do not even intimate that the raw products are probably contaminated when bought and thence how you should protect yourself from them. In commercial food establishments there have been drastic changes in the retail work force with extremely high turnovers. A less stable force means less training and knowledge about how to safely handle foods creating greater potential in restaurants, etc.

What has allowed all this to happen? Most medics have over the years become blasé about food-borne diseases. The American Public Health Association essentially ceased having scientific programs on food many years ago as did other groups. The 2nd National Conference on Food Protection in 1984 wasn't even going to have a microbiology section. This area has received a lower priority in funding as years went by. CDC reduced its efforts, Maine's sunset law was allowed to "wipe out" its food sanitation laws and Utah has proposed doing the same. Equally important, if not most, the bureaucratic responsibilities for foods at city-county-state, federal and international levels have become so intertwined with meshed responsibilities among different agencies that no one is in charge. With the result that even though we've

known about the growing problem with Salmonella since the 1950's, after each big outbreak that gets publicity the "committee effect" ensues. There's an investigation, the facts are usually obtained and then everyone sits around self-flagellating but does nothing due to individual interests or fear of stepping on territorial turf.

The USDA has played a part in this apathy also. They are by NO means the major culprit and indeed have done many things right, just not all the right things. They have very effectively removed diseased animals from the food chain and they have very effectively protected the safety of cured, cooked or ready-to-eat products. They have performed excellent studies in all phases of speeding up production, many areas of fraud protection for the consumer such as too much water, proper labels, etc. They have used great scientific knowledge to develop species tests so we now know whether chicken meat is in our beef hotdog. They however have always considered as "normal contaminants" those pathogens which produced no signs of clinical disease in the carcass or those accidentally placed on the carcass from the animal's own feces and hence these normals were not controllable. There has been no prodding to do otherwise from the administrations, congress nor industry. When urged and funded they began a residue control program. Residues (even though little disease is documented) is unknown and scary. Food-borne disease is well documented, known and not scary. There is now a

push to stop the old, old, organoleptic program and go to a new scientific program. Why not continue both, after all, gross pathology in humans or in animals is organoleptic by definition. It is the medically scientific way of seeing, feeling and diagnosing disease from medical signs. Even today it is still "scientific." No one even knows what is causing all the condemnations from the scientific organoleptic inspection which USDA has done so well for many decades. Is all this condemned diseased meat safe for humans (even though aesthetically repulsive)? Is all of it a public health threat? Is it 50% safe? Until this is known a judgement can't be made as to relinquishing control of pathology in addition to adding new "scientific" microbial tests.

In conclusion, each year we have a public health problem causing millions of cases of disease, hundreds of thousands of hospitalizations, thousands of deaths, thousands of deformed children, hundreds of thousands of chronic joint, nutritional, enteric or worse diseases. This is a problem from all meats, poultry and seafoods (poultry is probably not even the major problem). This is not a Salmonella problem and not just a fecal problem. There are many diseases you've heard about today, some are in the gut, some are in the muscle you eat. This relates to many industries and agencies (USDA is certainly not the only major player in this game). We currently spend about $1.60 per American per year on inspecting all meat and poultry slaughtered,

processed, imported into or exported from the U.S. That is an amazing cost benefit ratio and for another 4 or 5 cents we could really fund some good research aimed only at diseases and their control. We must change our attitude about quality control from being how good something looks to safety and quality assurance which determines how it will perform safely, nutritionally and aesthetically for the consumer.

You've heard a lot today about diseases. Some will say there's little that can be done. Others believe much can be done now and for little cost. We must start now, now is already too late. I believe we have the know-how to make a major dent in reducing these diseases immediately. We could ask Sweden, if we need help, since they have essentially eradicated Salmonella from their food supply.

Senator MELCHER. Dr. Menning, we talked earlier about the number of inspectors going down, and that each inspector has to look at more pounds of product, whether it is poultry or meat, per hour.

How serious is this?

Dr. MENNING. My personal opinion is that it is not very serious. I personally believe that the number of persons involved in inspecting is not as important today as a change in direction of how the inspection is done.

Senator MELCHER. Then it is the quality of inspection that is a problem?

Dr. MENNING. It would be a quality of inspection, if one means by that involving newer scientific tests or studies, and specific controls to control the diseases we have been talking about.

The inspection process, as I mention in my prepared statement, I believe USDA has been doing an outstanding job of removing diseased animals, or anything that looks aberrant.

What they have not been doing, and primarily because they have not been charged or urged to really, is look for specific disease organisms that are other than in the diseased animals that they condemn. This must be changed because we must continue to condemn the obviously diseased animals in addition, then, to trying to find some information on those good-looking animals, poultry or beef, that contain disease germs.

Senator MELCHER. Let's start on the premise you have just enunciated. We are looking at a healthy steer, or a healthy looking bird, and we will take it through the process.

Now in the case of beef, after it has been killed and skinned, and eviscerated, where is the salmonella coming from on the beef?

Dr. MENNING. Well, it could come from many sources. The primary source, however, would have been accidental contamination from fecal content from those beef.

Senator MELCHER. From the feces?

Dr. MENNING. That is correct.

Senator MELCHER. And so beef is not dipped. What are you going to recommend? Just wash it good?

Dr. MENNING. There are various studies that are going on as far as possibly finding that lactic acid, or acetic acid sprays may help reduce it. However, there are also various other things to do along the way, and that is to try and place more emphasis on what is known as the critical control points that may be causing more feces to get on the beef and/or to be further spread around by techniques of design, whatever it may be. So you have to determine what is on it, and where it gets on it, and then try and control it at those points.

Senator MELCHER. Then you are speaking of two things. You are speaking of the plant itself, and how clean it is, and how clean the animal is after it has been eviscerated and exposed to possible fecal contamination. Is that correct?

Dr. MENNING. Yes, sir.

Senator MELCHER. And if I understand your comments right, Dr. Menning, you have said that those matters can be, if not eliminated, at least minimized?

Dr. MENNING. They certainly are not going to be eliminated just within a plant. You have to start all the way back to the animal feed for that. But yes, the various stages can be caused to minimize the contamination.

Senator MELCHER. In the case of poultry, after they have been plucked, and after they have been eviscerated, are they not dipped then in a solution?

Dr. MENNING. They are washed, they are sprayed on the line, normally, and then are put into a chill tank of water. If this is what you are referring to as dipping.

Senator MELCHER. At what stage?

Dr. MENNING. Just at the end.

Senator MELCHER. At the very end.

Dr. MENNING. After they have been passed. The purpose is to chill their body temperature, which is high, as low as they quickly can, prior to putting it into a refrigerated room.

Senator MELCHER. Where is the salmonella controlled, then?

Dr. MENNING. Well, essentially, there is no salmonella control presently. Are you asking me where I believe the salmonella could be controlled?

Senator MELCHER. No, but that would be the next question.

Dr. MENNING. For all practical purposes there is no salmonella control program.

Senator MELCHER. At present?

Dr. MENNING. At present.

Senator MELCHER. The dipping is only for the purpose of chilling, and is not going to kill salmonella?

Dr. MENNING. If there is chlorine in it, it will kill a small percentage of any bacteria that happens to be present, but its purpose there is not specifically to reduce salmonella.

Senator MELCHER. Then the next question is your question. Where would you put the control?

Dr. MENNING. This gets very complex. One has to start at the farm. You have to start with assuring that salmonella-free, or other disease-free, disease germ-free feeds are fed to the poultry. I would also start a flock testing program on a statistical basis. You will then be able to tell which flocks may or may not have salmonella-free. You can then slaughter the salmonella-free birds, of which we have a fair amount of large flocks that are free of salmonella—slaughter them first in the day in the same plant; the salmonella-positive ones later in the day.

I would also attempt to try to figure out some way of changing the scald tank that heats them up to loosen their feathers because it is not hot enough. And you will excuse me, but, it is literally fecal soup that they are pulled through at about 126 degrees.

If you increase the temperature it is going to change the color of, or the bloom of, the bird, which the industry wants, and they think that people want. But it could be done. They could also be heated other ways. We might even have to think about skinning birds and not having to take the feathers off.

This would remove a lot of the fat anyway, but it would change habits. From there, then, is the picking machine which most people concede is one of the biggest contributors of adding salmonella into

the skin or on the skin. So, processes would have to be changed there, and there are ideas, but these all take a lot of time to state.

Senator MELCHER. Just to cap it up, Doctor, what you are recommending is that the birds be salmonella-free, and they can well be salmonella-free before they reach the plant itself?

Dr. MENNING. Yes, sir. Now this would be an ultimate goal. This is not something that is going to be done by a law that is passed, we will say, in 5 months, in ordering salmonella-free birds, you know, suddenly. It is a total program whose ultimate goal is that.

Senator MELCHER. Secondly, if they are not salmonella-free when they come to the plant, then the change should occur right at the moment of starting the plucking process?

Dr. MENNING. That is correct.

Senator MELCHER. To put it in an understandable way for everybody, when the bird is put in the vat, prior to plucking, feces are in that vat and are going to be in that vat, and they are going to contain salmonella unless they were salmonella-free before they got to the plant?

Dr. MENNING. Yes, sir.

Senator MELCHER. So therefore, it is at that point that you see the greatest danger for allowing salmonella to remain on the finished product?

Dr. MENNING. That is one of the major points; yes, sir.

Senator MELCHER. It is going to contaminate the skin, and it is not going to be removed at any point further on down the line?

Dr. MENNING. Not to any great degree, under present methods.

Senator MELCHER. What would all this improvement cost per pound? We will exclude the part about having them salmonella-free at the farm, because that is a different economic consideration. But at the plant itself, what would these improvements cost, changing the process to just as you have described, per pound?

Dr. MENNING. I have no idea, sir.

Senator MELCHER. Would it be 1 cent a pound?

Dr. MENNING. I would really hate to guess because I have nothing on which to base it. A personal wild guess would be that it would be no more than that, if that, but that is based on no knowledge of facts of cost.

Senator MELCHER. It very likely would be no more than 1 cent a pound. I will pay it. I think everybody else would pay it. And I think that the bloom on the skin, while it might be a selling point at the poultry counter in our supermarkets, I believe that it could be well sacrificed, but it would be sacrificed uniformly, if this were required, because everybody would have to put up with this type of inspection.

That is the beauty of Federal inspection. Once you have set the standards, then every plant has to follow.

Dr. Menning, why haven't these standards been set?

Dr. MENNING. I would only be able to guess, and that is they have not been set because various interests have not encouraged them to be, as well as other interests have discouraged them.

Senator MELCHER. Who should take the lead, Congress or the Department of Agriculture, or both?

Dr. MENNING. I think both.

Senator MELCHER. I am for that. I find it dismal, that the Department of Agriculture has not taken the lead on this, and I think that all of this sort of hemming and hawing on how much we can cut out in the cost of inspection is absolutely ridiculous. After all, what we provide on our tables for our families is the most critical thing that we do, daily, to make sure that it is healthy and does not cause us to get sick.

Thank you very much, Dr. Menning. I do want to get back to the other two doctors, but I will wait until after my colleague here, Senator Pryor, has had an opportunity to make his comments and ask his questions.

Senator PRYOR. Dr. Menning, if we adopted the Menning Plan or the Menning theory of salmonella elimination, would we actually eliminate salmonella, or further reduce it?

Dr. MENNING. Obviously you would not eliminate it for a long, long time, if ever, with just what I have mentioned here. There are various ways of course to go about—a great length of time to eliminate it.

Senator PRYOR. Isn't there only one true way—and let's be very honest—to totally eliminate salmonella, and that the only way that has ever been suggested as a final option is through a system we would call irradiation, or I guess you would say "nuking" the product. Is that correct? That would eliminate salmonella, is that correct?

Dr. MENNING. It could eliminate salmonella if there were high enough——

Senator PRYOR. But it may cause problems even greater than salmonella, would this be correct?

Dr. MENNING. Some persons feel it would. I have no knowledge, up to a certain level, that it would cause any problems.

Senator PRYOR. I think the line of questioning of Senator Melcher was very helpful and constructive about the process of poultry, about the process of beef, and where we should make the change. I think we are really getting down, now, to an area that all of us should cooperate in.

I would like to add one more point, Senator Melcher, about the Department of Agriculture or the Food and Drug Administration, or the Congress, or whoever, taking the lead.

I think the industry has got to take a lead, and I can say, without reservation, that the industry is willing to be very supportive in any program that is going to increase the safety of food consumption. I cannot speak for all industries, but I hope in this area I can speak for the poultry industry.

Now, I think we ought to get one other point in the record, and I am not sure of the answer. When an inspector touches a carcass of beef, or touches one of the many hundreds or thousands of birds a day going through the line, can that inspector, by merely touching that bird or carcass, actually contaminate that bird, or that beef, or swine?

Dr. MENNING. Certainly. Hands.

Senator PRYOR. In other words, there can be crosscontamination by the inspectors?

Dr. MENNING. Hands are the biggest contaminators of foodborne disease, especially in the home or in industry.

Senator PRYOR. All right. I also think for the record we need to get a point cleared up on the so-called chill tank. Senator Bumpers, earlier this morning, made reference to a CBS documentary, I believe it was on "60 Minutes," and the indication, or the implication, was that the chill tank was the last process before the bird became packaged.

But isn't it true, that after the chill tank the bird is once again washed?

Dr. MENNING. In some plants.

Senator PRYOR. Is there any question that all the plants wash after the chill tank? I do not know. If they do not, they should.

Dr. MENNING. I have no knowledge that all plants do wash after the chill tank.

Senator PRYOR. I am almost certain, that after the chill tank there is another washing of the bird.

Well, do you want to ask other questions, Senator Melcher? If I might ask Dr. Archer a question. How many inspectors does the Food and Drug Administration have?

Dr. ARCHER. I am unsure of the answer to that, sir. That is not in my purview.

Senator PRYOR. I am not trying to supply you with the answer but I would suggest in the neighborhood of 400. I think that would be roughly correct.

And how many plants do you have jurisdiction over?

Dr. ARCHER. That, too, again, is the compliance aspect of the Agency and I am not a part of that function, sir.

Senator PRYOR. Well I think the number there, if I might suggest again, is 6,000.

Now, would you mind comparing, just for a moment, the different ways, or the different methods FDA uses in inspecting a product compared to the Department of Agriculture. Do you have a sampling technique?

Dr. ARCHER. Again, Senator, I am not sure of the domestic inspection routines as the compliance end of the Agency does it. I could only speak to what we might do within our area on import samples, which is the only thing I really have familiarity with.

Senator PRYOR. Are you aware of any inspection program in FDA for seafood and for fish?

Dr. ARCHER. No, sir. I am aware of the cooperative relationship we have between the producing States in the form of the Interstate Shellfish Sanitation Commission.

Senator PRYOR. Do you think that program needs to be reinforced, either with money or new inspection programs, or new bodies, or what?

Dr. ARCHER. The nature of the problem, to underscore what Dr. Cohen said earlier, resides more in the area of shellfish than fin fish and the particular problems concern the viral diseases—and what we need is a rapid advance in technology.

At the present time, the technology to prevent or even detect many of the diseases that are transmissible by shellfish is simply not existent.

Senator PRYOR. Dr. Roberts, if I might ask you what you mean when you use this word. I am just a layman in all of this. What do you mean when you say subjective assumptions?

Dr. ROBERTS. When you are putting together the cost data, you have to make a number of assumptions as you are moving along. You have to figure out how many cases are going to be called mildly severe, what data bases you are going to use for the moderately severe cases, where you are going to get your data for deaths, and how you are going to put the pieces together.

Senator PRYOR. And by the way, I compliment you. I know that all of this data must be very hard to come by, and you are using many reports and many studies, and many facts and figures.

What would you say that the error rate is? For instance, in a poll by ABC Nightly News or someone, they will state the error rate may be 2 or 3 percent on one side or another.

Is there an error rate that you have established for yourself, or is there an error rate you have established in trying to assimilate and produce this data?

Dr. ROBERTS. Well, the reason I used both a high and a low estimate is to give people the idea that there is a range involved, and, frankly, I do not know what the error is.

Senator PRYOR. I see.

Dr. ROBERTS. I try to be as accurate as possible in the use of these numbers, but I do not know for sure.

Senator PRYOR. For the time being, that is all. Thank you, Senator Melcher. Thank you.

Senator MELCHER. Dr. Archer, do you have figures that you can share us with regarding arthritis as a result of either salmonellosis, or an infection from campylobacter?

Dr. ARCHER. Yes, and again the figures are estimates, not my estimates, but they are based on a compilation of clinical literature. Most of the time they are hindsight studies. That is, the outbreak has occurred, the people have been ill, and later, only physicians who are interested in this particular sequela to the acute infection may report out of their own interest group, such as rheumatologists in the case of rheumatoid diseases.

There is a syndrome that can follow infection with either salmonellosis or salmonella, or campylobacter, which is known as reactive arthritis. It is a painful disease in the same sense that rheumatoid arthritis would be, but there are many significant differences, the main one being that, by and large, most people spontaneously recover from reactive arthritis within a given time period, usually within 6 months. And in very rare cases it may go on more than 1 year.

They do require medication, however, and it is certainly a disconcerting condition. The estimates vary by the causative organism, but a safe range and a good guess would be about 1 to 3 percent, based on the bulk of the clinical literature, and based on a small survey that was done following the Chicago milk outbreak.

Senator MELCHER. Isn't it also true that heart disease can follow colitis?

Dr. ARCHER. There are many different, more serious conditions that can come about as a result of an acute foodborne infection, that is true. It has to be put in the context that these numerically, are rare. Obviously, to the individual infected, that does not mean much.

In the case of salmonella, for instance, we have to look at it not in the case of the high-side estimate, say we are working with a 2 million total infected population estimate. We would have to look at the CDC figures for actual reported cases, because those are the ones that probably sought medical attention, and work with that lower figure, say, 40 to 50,000 cases.

In that situation, a good estimate is that somewhere between 5 and 10 percent may develop septicemia, or blood infection which will require hospitalization, and some proportion of that, which is even smaller, may develop focal infections anywhere, including the meninges, resulting in meningitis.

Senator MELCHER. Dr. Roberts, I do not believe you have taken into consideration in your economic study anything more than the first impact of foodborne diseases, isn't that true?

Dr. ROBERTS. Yes, that is true. I have not included these chronic complications that Dr. Archer mentioned. I also have not included the psychic costs of time lost from other nonwork related activities, or cost to industry, or public health surveillance costs.

Senator MELCHER. So, Dr. Menning, we finally get back to you again, and, the fact is that the little bit of cost involved is so infinitesimal, as compared to the loss that is involved, and so whatever it takes, whatever has to be done ought to be done, not just for economic reasons but because we want to be more comfortable in reassuring ourselves that we have done the utmost.

I want to commend all of you. I think these are very good papers. Dr. Roberts, I am proud to see that the Department of Agriculture does have economists that—and I am not deploring any of the economists you have—but I am proud to see that some of them actually zero in on costs related to people directly in their health matters.

Dr. ROBERTS. Thank you, Senator.

Senator MELCHER. I want to thank you personally for being here, but I think we might have some more questions and some more comments from this great doctor here.

Senator PRYOR. Dr. Pryor here.

Senator MELCHER. Dr. Pryor.

Senator PRYOR. We are all becoming microbiologists here, lately. No, Mr. Chairman, I have no more questions. I want to express my gratitude to this panel. I also say to you, Senator Melcher, and to Chairman Leahy, I think it is very timely that he called this particular hearing at this particular time.

I think that this has been not only constructive, but I think that beyond that, it has been educational, educational for members of this committee, and educational for all of us as the consuming public, and I hope that what we have learned today can be translated, ultimately, into something very positive, and something that will be very beneficial to the consumers of this country, and we are very indebted to all of you, and to the preceding panelists who came before you. Senator Melcher, I thank you, and once again, I thank the panel.

Senator MELCHER. Thank you. The committee is adjourned.

[Whereupon, at 12:10 p.m., the committee adjourned, subject to the call of the Chair.]

[Material submitted for the record follows:]

June 16, 1987

Senator Patrick Leahy
Chairman of Agriculture, Nutrition,
and Forestry Committee
Attn: Chief Clerk Sarcone
Suite SR-328A Russell Building
Washington, DC 20510

Dear Senator Leahy:

The American Association of Retired Persons (AARP), a membership organization of more than 25 million Americans aged 50 and over, respectfully submits these comments for the record concerning the safety and wholesomeness of poultry products, and the National Research Council's Report. "Poultry Inspection, the Basis for a Risk-Assessment Approach".

The National Academy of Sciences in its May 12, 1987 report to USDA said the government's system of visual and manual inspections "does very little to protect the public against microbial hazards in boiler chickens because bacterial and chemical adulteration cannot be seen". The study also concluded that contamination with salmonella or other microorganisms is the most significant public health risk posed by consumption of chickens. AARP wishes to emphasize the impact of foodborne illnesses on older persons and to comment on two aspects of the report, the recommendation to implement new procedures and the labeling of poultry to prevent illness.

FOOD-BORNE ILLNESSES AND OLDER PERSONS

The fact that salmonella is a serious health threat has been well documented. In 1970 the National Research Council estimated that there were 2,000,000 cases of salmonellosis per year in the United States. Today, health authorities estimate that this figure has increased to 4,000,000 cases. Most victims of this disease dismiss the symptoms as a cold, virus or stomach ache and therefore never enter a hospital. Reported figures, then, are probably only a fraction of the cases of salmonellosis that actually occur.

AARP's concern is that medical authorities are seeing more severe cases of salmonellosis and even fatalities in older persons. Reports indicate that there are between 4,500 and 9,000 deaths that occur each year due to foodborne infections. While there are no national figures available on mortality rates among the aged, there is evidence that these illnesses, which cause discomfort for younger, stronger people can inflict severe suffering and death on frail older persons. Frequently suffering is intensified due to

mental and/or physical impairment.

Older persons, especially those who are frail, are often unable to detect potential hazards in contaminated food due to lessening sensory capabilities. Many may not be alert to food with undesirable appearance, extraneous matter or color changes. Therefore an older person might eat something a younger person would reject as unfit for consumption.

Foodborne illness outbreaks are especially dangerous in nursing homes. In May 1984 a salmonella outbreak in a Texas nursing home killed one older person and hospitalized 13 others. A similar outbreak in a Connecticut nursing home in 1986 led to the deaths of five elderly patients between 77 and 96 years of age. These occurrences point to a need to educate nursing home food service management and other food handlers who are responsible for proper food handling and preparation for vulnerable segments of the population. They also stress the importance of an across-the-board approach to effective inspection and regulation by state and local government as well as federal agencies.

The National Research Council Report

AARP supports the findings of the report that the federal government's inspection system is no longer sufficient to detect modern health dangers. It is our belief, however, that the committee's recommendation to shift emphasis from the current bird-by-bird inspection procedures to a system of random sampling of chickens for microbial and chemical contaminants is seriously flawed. Random sampling for such contaminants cannot be performed on site and results are not known before the bird is packaged, sent to the market and eaten. There is nothing to prevent contaminated chickens from ending up on the plate of an unknowing consumer.

The Association supports attempts to increase the efficiency of inspections. While random sampling is a good scientific approach, it is not a practical alternative for consumers counting on a safe food supply. Until more effective procedures are developed, we recommend that USDA return to the Poultry Inspection regulation changed in 1978, Part 381.91(b), which not only calls for visual and manual inspection but the condemning of all poultry carcasses found to be adulterated by filth and fecal matter.

The National Research Council's report has also suggested that labels be attached to poultry "to remind consumers about the preparation procedures necessary to avoid illness". AARP agrees that consumer information is essential and product labeling should be explored. We also encourage the expansion of educational efforts for all food handlers.

In summary, it is AARP's hope that before there is any significant shift in the inspection system: (1) there be adequate

tests to assure that poultry is free of contamination, (2) adulterated poultry be condemned so it does not end up in the marketplace, (3) on-going efforts be made to expand education to consumers and food handlers in proper handling and preparation, and (4) any new inspection system be thoroughly evaluated to ensure that it is better than the one we are replacing.

Sincerely,

John C Rother

John C. Rother
Director
Legislation, Research and
Public Policy